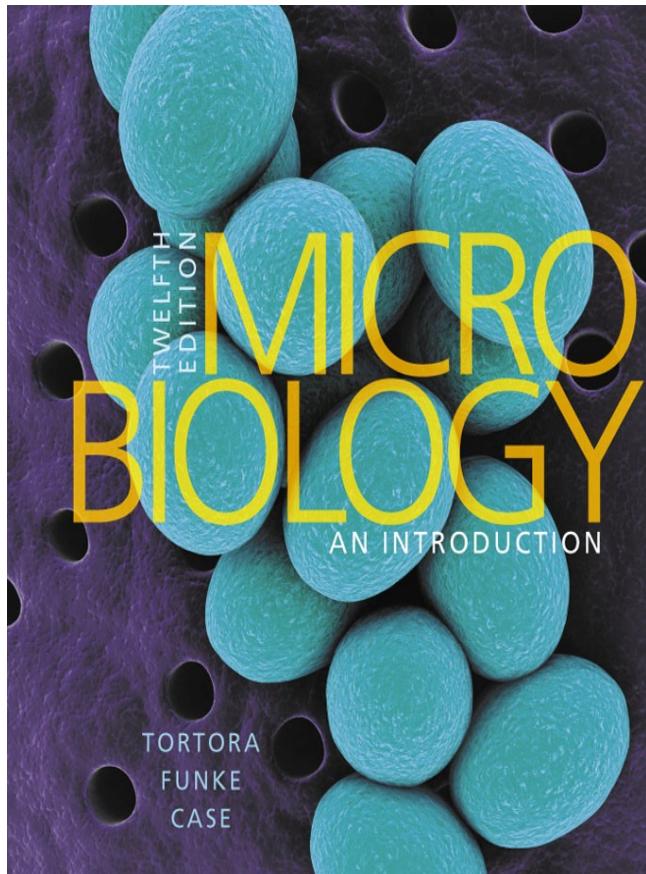


Microbiology an Introduction

Twelfth Edition



Chapter 25

Microbial Diseases of the Digestive System

Escherichia Coli Bacteria



Structure and Function of the Digestive System (1 of 3)

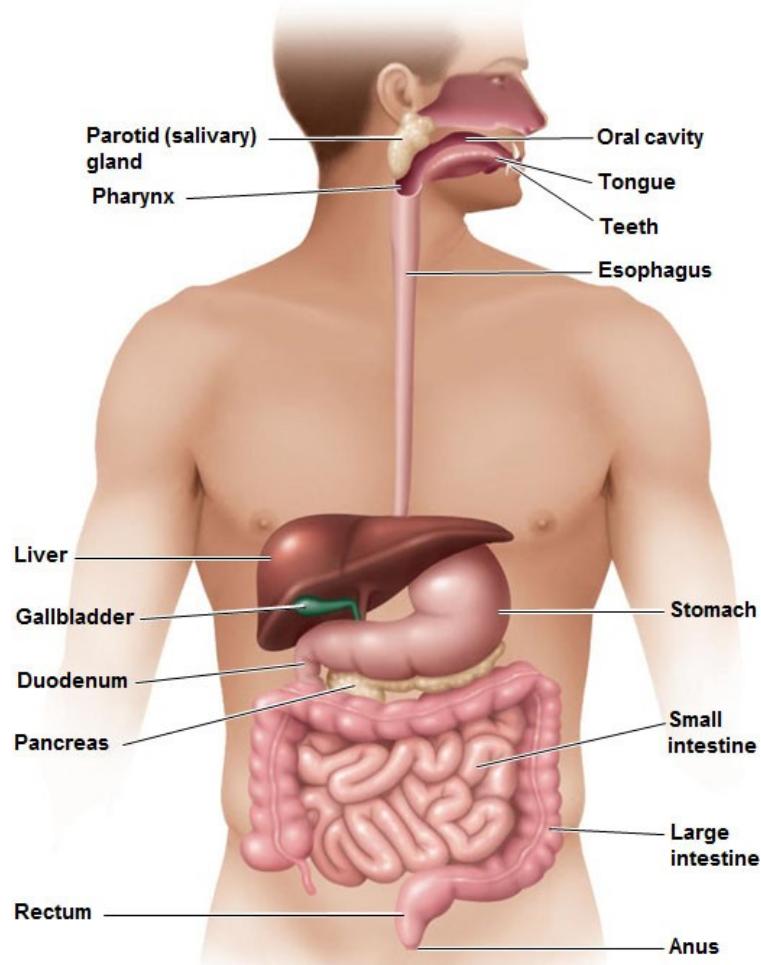
Learning Objective

25-1 Name the structures of the digestive system that contact food.

Structure and Function of the Digestive System (2 of 3)

- Gastrointestinal (GI) tract or alimentary canal
 - Mouth
 - Pharynx (throat)
 - Esophagus
 - Stomach
 - Small and large intestine
- Accessory structures
 - Teeth and tongue
 - Salivary glands
 - Liver
 - Gallbladder
 - Pancreas

Figure 25.1 The human digestive system



Structure and Function of the Digestive System (3 of 3)

- Absorption of foods
 - 25 tons of food pass through the GI tract in a lifetime
- 80% of the immune system is located in the intestinal tract
 - Gut-associated lymphoid tissue (GALT)
 - Lymph nodes
 - Peyer's patches

Check Your Understanding-1

Check Your Understanding

- ✓ Small explosions have occurred when a surgeon used spark-producing instruments to remove intestinal polyps. What ignited?

25-1

Normal Microbiota of the Digestive System (1 of 2)

Learning Objective

25-2 Identify parts of the gastrointestinal tract that normally have microbiota.

Normal Microbiota of the Digestive System (2 of 2)

- Millions of bacteria per ml of saliva
- Few microorganisms in the stomach
 - Due to HCl production
- Small intestine
 - Paneth cells
 - Granule-filled phagocytic cells; produce defensins
- Large numbers of bacteria in the large intestine
 - Anaerobes and facultative anaerobes
- 100 billion bacteria per gram of feces

Check Your Understanding-2

Check Your Understanding

- ✓ How are normal microbiota confined to the mouth and large intestine?

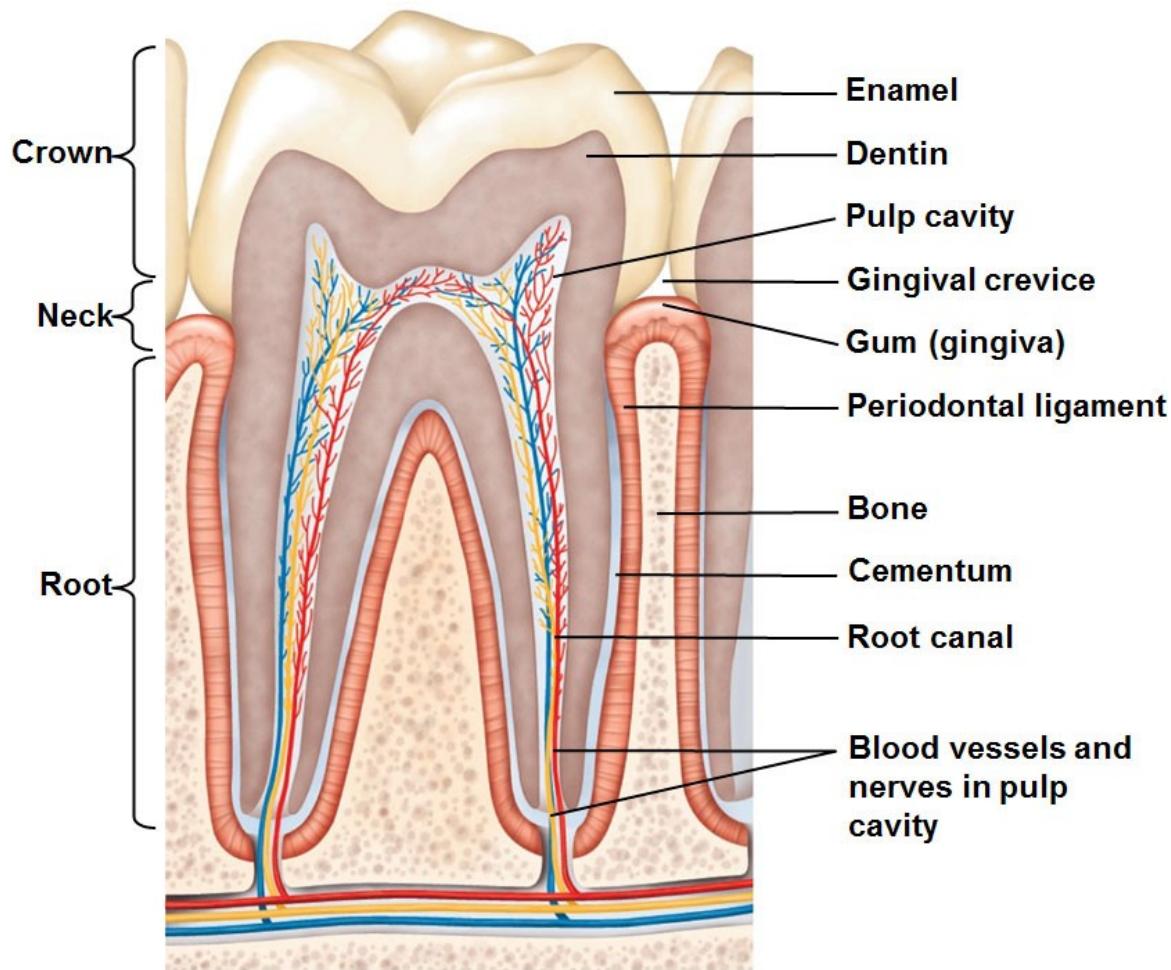
25-2

Bacterial Diseases of the Mouth

Learning Objective

25-3 Describe the events that lead to dental caries and periodontal disease.

Figure 25.2 A healthy human tooth



Dental Caries (Tooth Decay) (1 of 2)

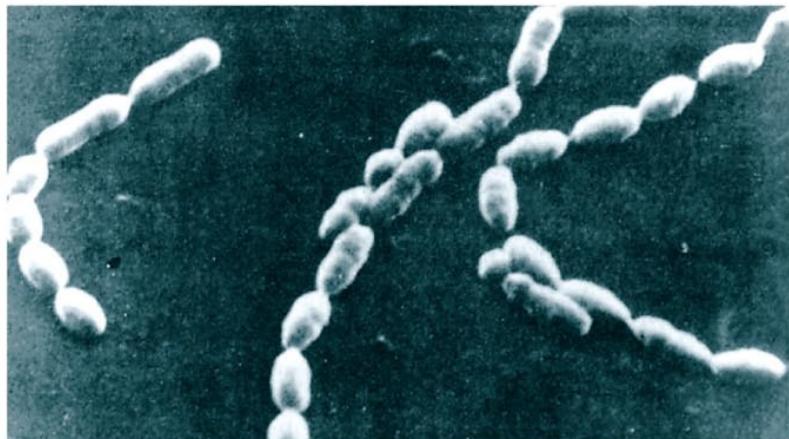
- **Dental plaque**
 - Biofilms involved in the formation of **dental caries**
- 700 species of bacteria in the oral cavity
 - **Streptococcus mutans** is the most important cariogenic organism
 - Gram-positive coccus
 - Converts sucrose to lactic acid
 - Produces dextran, a polysaccharide that forms plaque
- Plaques attract other cariogenic bacteria
 - Streptococci; filamentous **Actinomyces**



Pearson

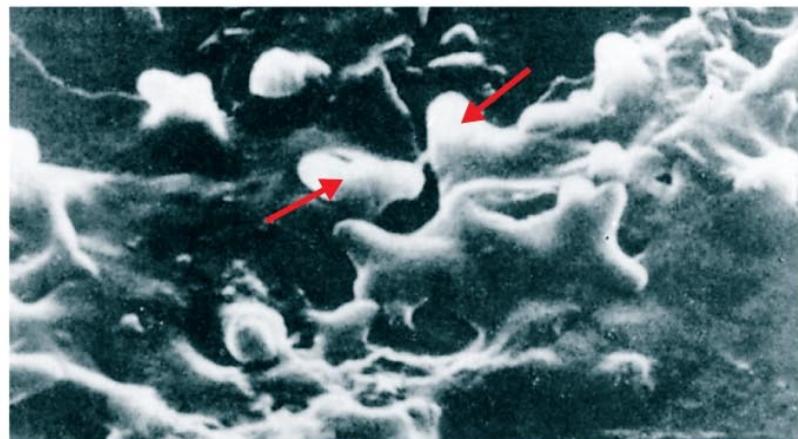
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Figure 25.3 *Streptococcus mutans*



(a) *S. mutans* growing in glucose broth

SEM
1 μm



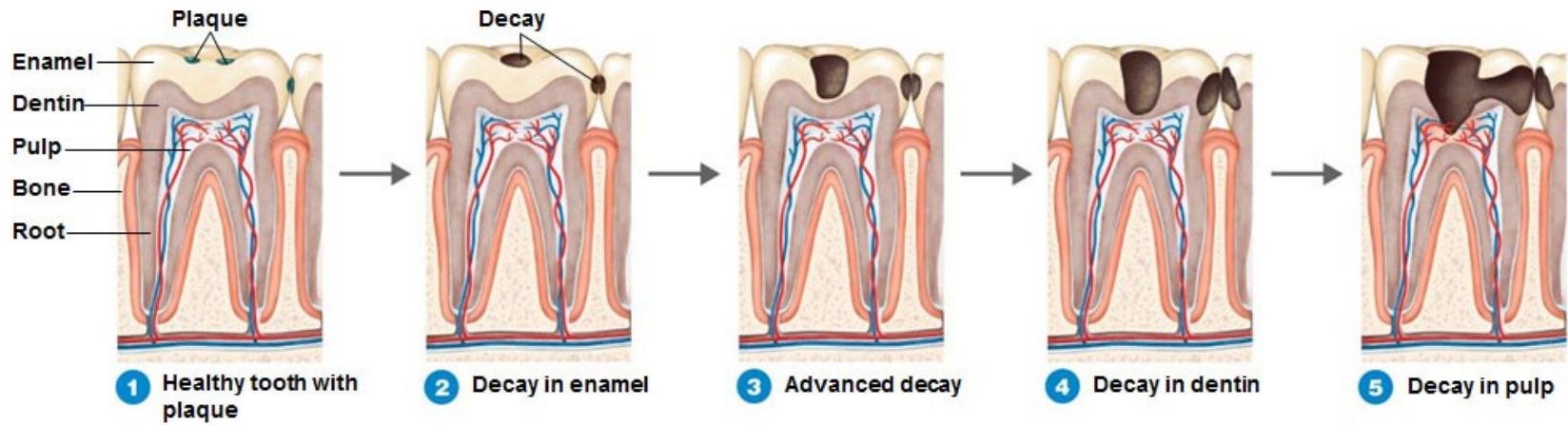
(b) *S. mutans* growing in sucrose broth; note the accumulations of dextran. Arrows point to *S. mutans* cells.

SEM
1 μm

Dental Caries (Tooth Decay) (2 of 2)

- Caries penetrate from enamel into the dentin
 - Caused by gram-positive rods and filamentous bacteria
- Decay can reach pulp, which contains the blood supply and nerve cells
 - May advance to the soft tissues, leading to abscesses
- Introduction of table sugar in the diet is correlated with the level of dental caries

Figure 25.4 The stages of tooth decay



Periodontal Disease

- **Gingivitis**

- Inflammation and infection of the gums
 - Caused by streptococci, actinomycetes, and anaerobic gram-negative bacteria

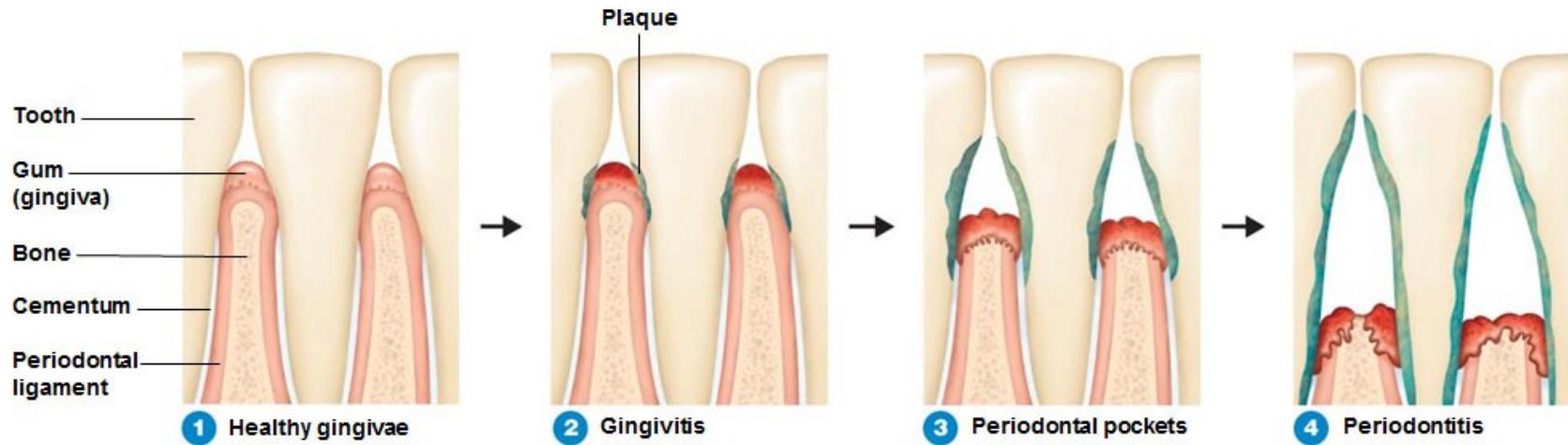
- **Periodontitis**

- Bone and tissue supporting the teeth are destroyed
 - Caused by **Porphyromonas**

- **Acute necrotizing ulcerative gingivitis (trench mouth)**

- Caused by **Prevotella intermedia**

Figure 25.5 The stages of periodontal disease



Diseases in Focus: Bacterial Diseases of the Mouth

- Can you identify infections that could cause persistent sore, swollen, red, or bleeding gums, as well as tooth pain or sensitivity and bad breath?

Diseases in Focus 25.1 (1 of 2)



Diseases in Focus 25.1 (2 of 2)

Disease	Pathogen	Symptoms	Treatment	Prevention
Dental Caries	Primarily Streptococcus mutans	Discoloration or hole in tooth enamel	Remove decayed area	Brushing, flossing, reducing dietary sucrose
Periodontal Disease	Various, primarily Porphyromonas spp.	Bleeding gums, pus pockets	Remove damaged area; antibiotics	Plaque removal
Acute Necrotizing Ulcerative Gingivitis	Prevotella intermedia	Pain chewing, halitosis	Remove damaged area; metronidazole	Brushing, flossing

Check Your Understanding-3

Check Your Understanding

- ✓ Why are "sugarless" candies and gum, which actually contain sugar alcohols, not considered cariogenic?

25-3

Bacterial Diseases of the Lower Digestive System

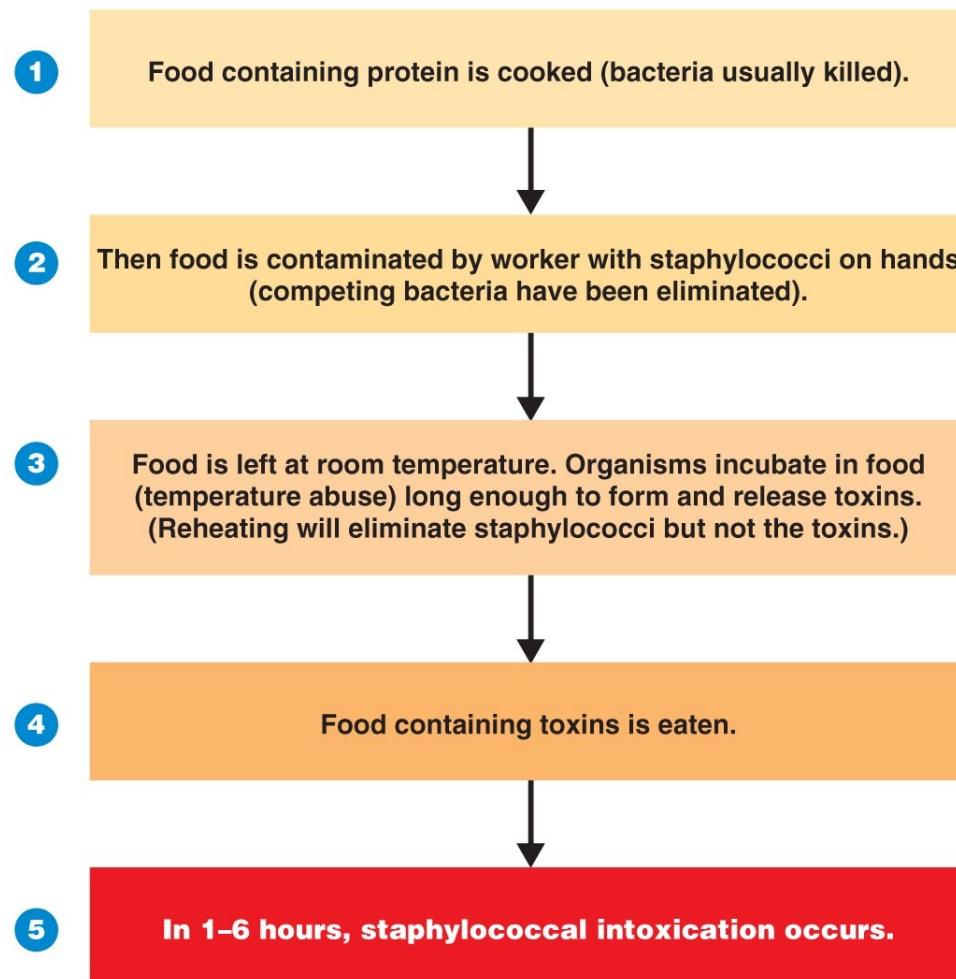
Learning Objective

25-4 List the causative agents, suspect foods, signs and symptoms, and treatments for staphylococcal food poisoning, shigellosis, salmonellosis, typhoid fever, cholera, gastroenteritis, and peptic ulcer disease.

Staphylococcal Food Poisoning (Staphylococcal Enterotoxicosis)

- Enterotoxin produced by **Staphylococcus aureus**
 - Serological type A
 - Coagulates blood plasma
 - Toxin is not killed by boiling
- Toxins produced when the organism is allowed to incubate in food (**temperature abuse**)
 - **S. aureus** outgrows most bacteria in high osmotic pressure and high temperature
- Phage typing traces sources of contamination

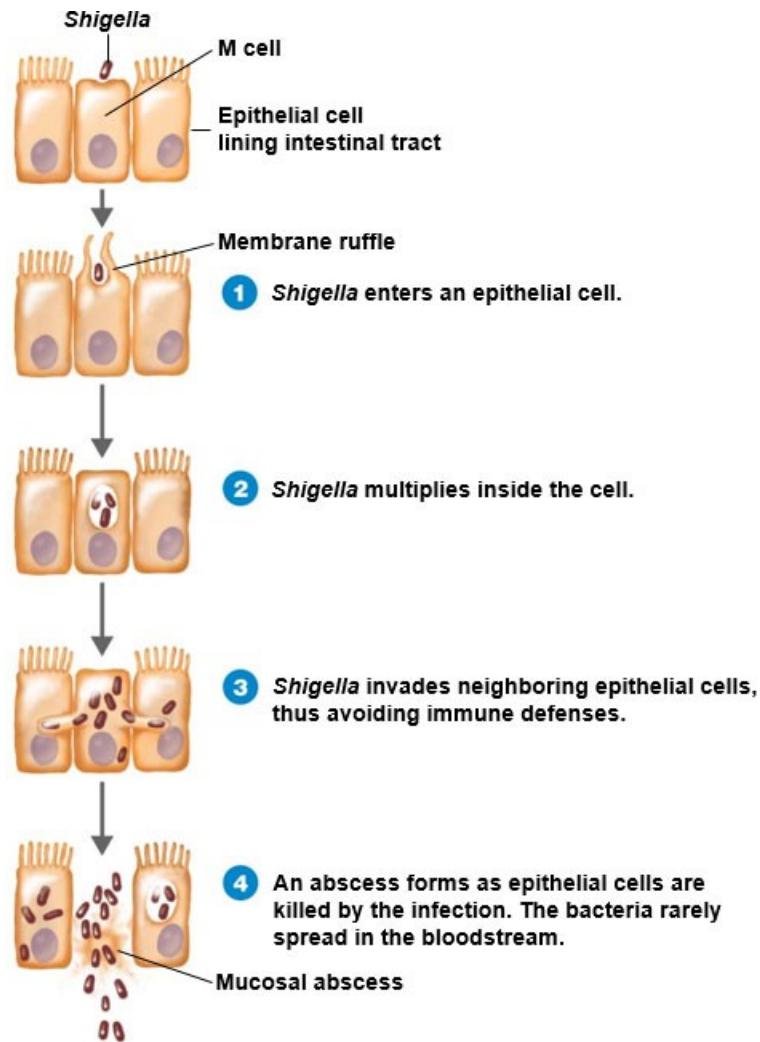
Figure 25.6 the Sequence of Events in a Typical Outbreak of Staphylococcal Food Poisoning



Shigellosis (Bacillary Dysentery)

- Caused by the genus **Shigella**
 - Facultatively anaerobic; gram-negative
 - Four species
- Produces the **Shiga toxin**
- Small infectious dose
 - Attaches to M cells, invades, and spreads to other cells
 - Damage to the intestinal wall
 - Can invade the bloodstream
- Treated with fluoroquinolones and oral rehydration

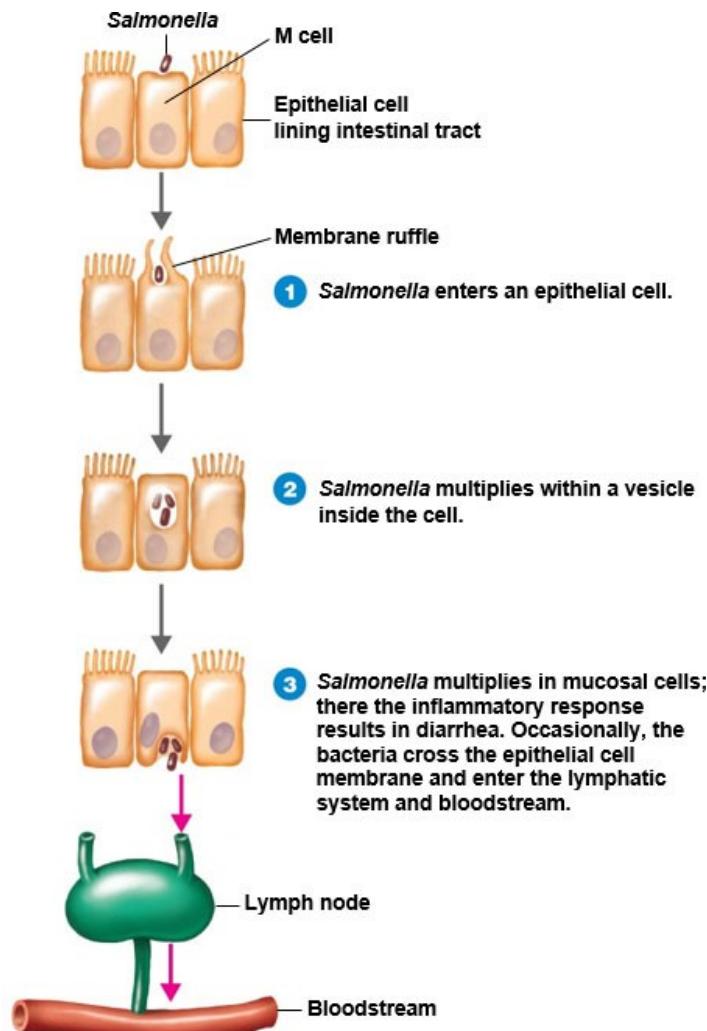
Figure 25.7 Shigellosis



Salmonellosis (Salmonella Gastroenteritis) (1 of 2)

- **Salmonella enterica**
 - Gram-negative, facultative anaerobe, nonendospore-forming rods
 - Normal inhabitant of the human intestinal tract
 - 2000 serotypes
- Invades intestinal mucosa and multiplies
 - Passes through M cells and enters the lymphatics
 - Replicates in macrophages
- Incubation of 12 to 36 hours; fever, nausea, pain and cramps, diarrhea

Figure 25.8 Salmonellosis



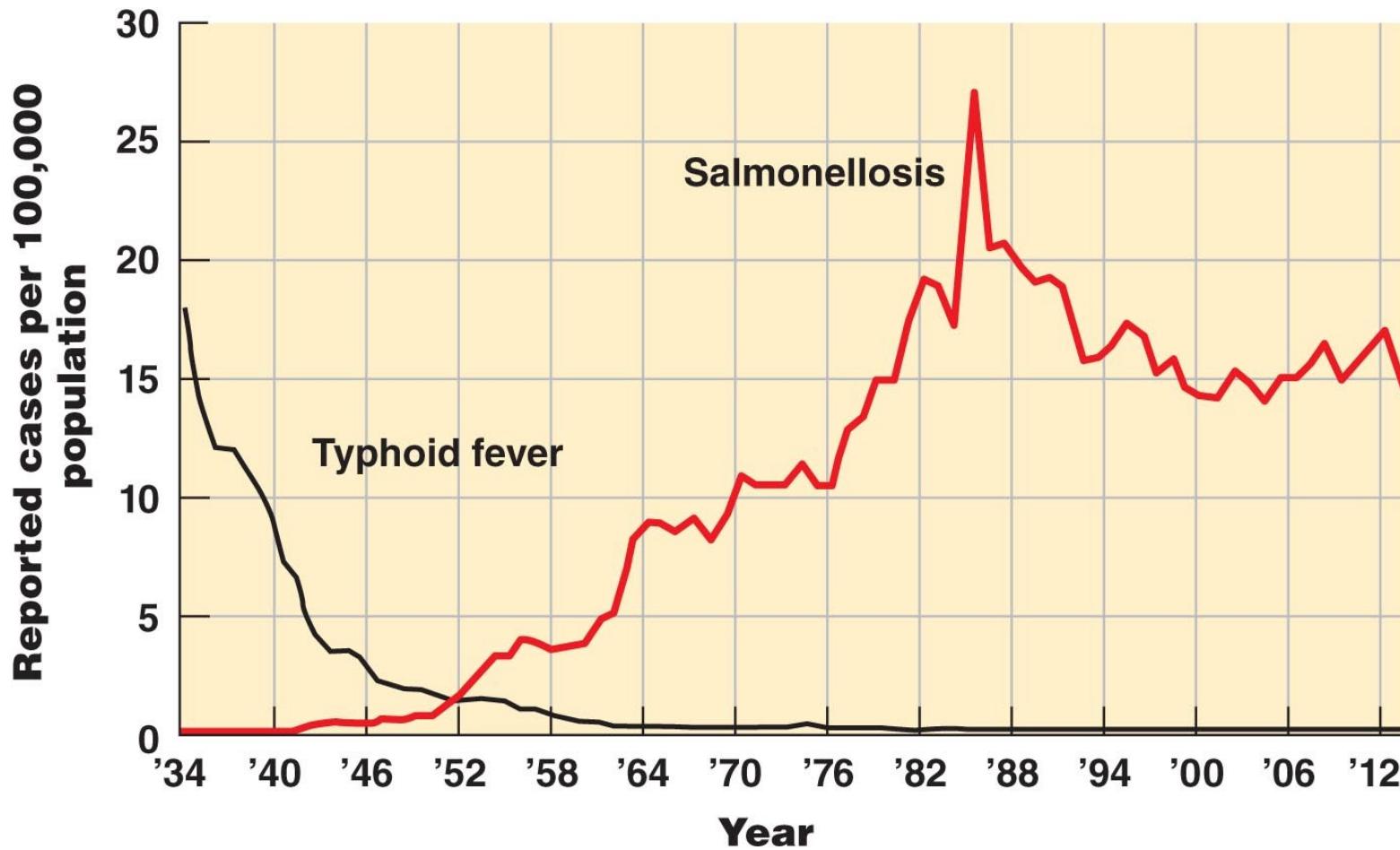
Salmonellosis (Salmonella Gastroenteritis) (2 of 2)

- 1.4 million cases; 400 deaths annually
- Associated with commercial chicken and egg production
 - Bacteria survive in the albumin
- Diagnosed directly from the stool or by PCR
- Treatment with oral rehydration therapy

Typhoid Fever

- Caused by **Salmonella typhi**
 - Spread only by human feces
 - Rare in the United States today due to sanitation
- Bacteria spread throughout the body in phagocytes
 - Releases organism into the bloodstream
- High fever, headache, intestinal wall ulceration
- 1-3% of patients become chronic carriers
 - Harbor the organism in the gallbladder
- Treated with chloramphenicol, quinolones, or cephalosporins

Figure 25.9 The Incidence of Salmonellosis and Typhoid Fever



Check Your Understanding-4

Check Your Understanding

- ✓ Why was typhoid fever almost entirely eliminated in developed countries by modern sewage treatment whereas salmonellosis has not been?
25-4

Cholera

- Caused by **Vibrio cholerae**
 - Slightly curved, gram-negative rod with single polar flagellum
 - Associated with salty waters
 - Produces the cholera toxin
 - Toxin causes the host cells to secrete electrolytes and water
 - Causes "rice water stools"
- Can lose 12 to 20 liters of fluid per day
 - Causes shock, collapse, organ failure, and death
- Treatment includes IV fluid replacement

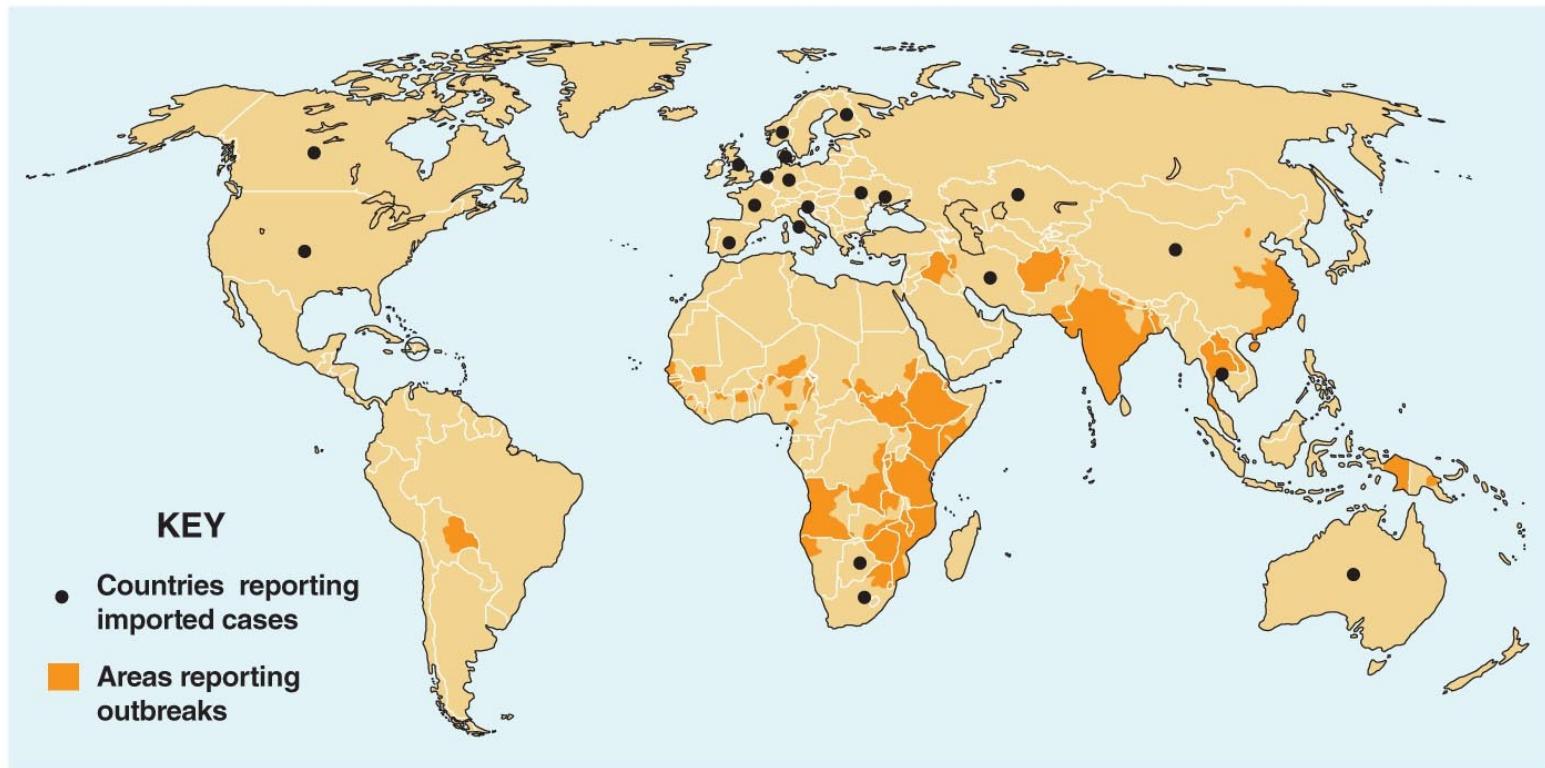
Figure 25.10 *Vibrio Cholerae*, the Cause of Cholera



Big Picture: Cholera after Natural Disasters (1 of 2)

- Cholera increases when sanitation and sewage disposal systems are compromised
- Outbreak in Haiti after earthquake due to deficient septic system at the Nepalese base
 - Nepalese soldiers were part of the United Nations peacekeeping force

Big Picture pg. 720



Big Picture: Cholera after Natural Disasters (2 of 2)

- Strategies for disaster preparedness
 - Oral rehydration solutions
 - Salt, sugar, and water
 - Stockpiling vaccines
- The ultimate solution
 - Proper sanitation, water storage, handwashing

Noncholera Vibrios

- Mostly adapted to salty coastal waters
 - **Vibrio parahaemolyticus**
 - Found in saltwater estuaries; common cause of gastroenteritis
 - Raw oysters and crustaceans are associated with outbreaks
- Require early antibiotic therapy

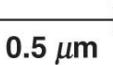
Escherichia coli Gastroenteritis

(1 of 2)

- **Enteropathogenic E. coli (EPEC)**
 - Diarrhea in developing countries
 - Cause the host cells to form pedestals where the bacteria attach
- **Enteroinvasive E. coli (EIEC)**
 - Causes Shigella-like dysentery
- **Enteropathogenic E. coli (EAEC)**
 - Only in humans
 - Produce an enterotoxin causing watery diarrhea

Figure 25.11 Pedestal formation by Enterohemorrhagic E. coli (EHEC) O157:H7



SEM  0.5 μm

Escherichia coli Gastroenteritis

(2 of 2)

- **Enterohemorrhagic E. coli (EHEC)**

- Produces Shiga-like toxin
 - Released upon the cell's lysis
- Most outbreaks are due to serotype O157:H7
- Cattle are the main reservoir
- Causes hemorrhagic colitis and hemolytic uremic syndrome
- Diagnosed by the inability to ferment sorbitol and pulsed-field gel electrophoresis

Traveler's Diarrhea

- Most common cause is ETEC; second most common is EAEC
- Can also be caused by **Salmonella**, **Shigella**, and **Campylobacter**
- Oral rehydration therapy and bismuth-containing preparations

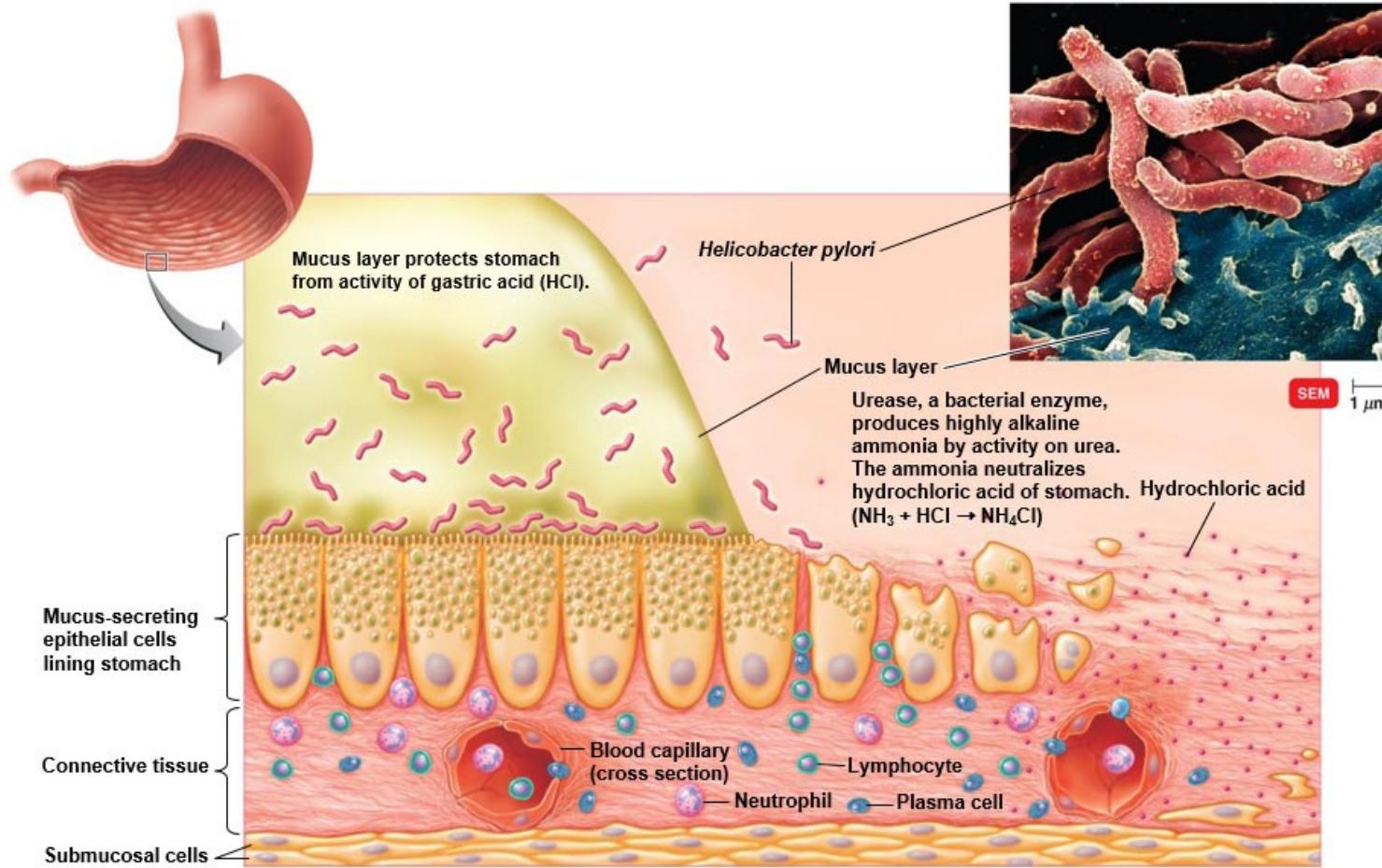
Campylobacter Gastroenteritis

- Caused by **Campylobacter jejuni**
 - Gram-negative, microaerophilic, spirally curved
- Leading cause of foodborne illness in United States
 - Common in the intestines of poultry
 - 60% of cattle excrete organism in feces and milk
- Fever, cramping, abdominal pain, diarrhea, dysentery
- 1 in 1000 cases leads to Guillain-Barré syndrome

Helicobacter Peptic Ulcer Disease

- Caused by **Helicobacter pylori**
 - Infects 30-50% of the population in the developed world
 - Grows in the stomach acid by producing urease
 - Converts urea to alkaline ammonia
 - Disrupts stomach mucosa, causing inflammation
- Treated with antimicrobial drugs and bismuth subsalicylate
- Diagnostic test requires a biopsy, culture, and urea breath test

Figure 25.12 Helicobacter Pylori Infection, Leading to Ulceration of the Stomach Wall



Yersinia Gastroenteritis

- Caused by ***Yersinia enterocolitica*** and ***Yersinia pseudotuberculosis***
 - Gram-negative
 - Inhabits animals; transmitted in meat and milk
 - Grows at 4°C
- Diarrhea, fever, headache, abdominal pain
 - Often misdiagnosed as appendicitis
- Treatment with antibiotics and oral rehydration

Clostridium perfringens

Gastroenteritis

- Gram-positive, endospore-forming, obligately anaerobic rod
- Also causes gas gangrene
- Associated with foods containing animal intestinal contents
 - Creates low oxygen level
- Produces an exotoxin
- Symptoms are usually mild
 - Occur 8 to 12 hours after ingestion

Clostridium difficile-Associated Diarrhea

- Gram-positive, endospore-forming anaerobe
- Causes more deaths than all other intestinal infections combined
 - Mostly in health care settings
- Life-threatening colitis
 - Ulceration and perforation of the intestinal wall
- Precipitated by the extended use of antibiotics
 - Eliminates competing intestinal bacteria

Bacillus cereus Gastroenteritis

- Large, gram-positive, endospore forming
 - Common in soil and vegetation
- Spores survive heating
 - Germinate and produce toxins
 - Different toxins cause different symptoms

Diseases in Focus: Bacterial Diseases of the Lower Digestive System

- An 8-year-old boy has diarrhea, chills, fever (39.3°C), abdominal cramps, and vomiting for 3 days. The next month, his 12-year-old brother experiences the same symptoms. Two weeks before the first patient became ill, the family had purchased a small (< 10 cm) red-eared slider turtle at a flea market.
- Can you identify infections that could cause these symptoms?

Diseases in Focus 25.2 (1 of 3)

Disease	Pathogen	Symptoms	Intoxication/ Infection	Diagnostic Test	Treatment
Staphylococcal Food Poisoning	<i>Staphylococcus aureus</i>	Nausea, vomiting, and diarrhea	Intoxication (enterotoxin)	Phage typing	None
Shigellosis (bacillary dysentery)	<i>Shigella spp.</i>	Tissue damage and dysentery	Infection (endotoxin and Shiga toxin, exotoxin)	Isolation of bacteria on selective media	Quinolones
Salmonellosis	<i>Salmonella enterica</i>	Nausea and diarrhea	Infection (endotoxin)	Isolation of bacteria on selective media, serotyping	Oral rehydration
Typhoid Fever	<i>Salmonella typhi</i>	High fever, significant mortality	Infection (endotoxin)	Infection (endotoxin) Isolation of bacteria on selective media, Serotyping	Quinolones; cephalosporins
Cholera	<i>Vibrio cholerae</i> O:1 and O:139	Diarrhea with large water loss	Infection (exotoxin)	Isolation of bacteria on selective media	Rehydration; doxycycline

Diseases in Focus 25.2 (2 of 3)

Disease	Pathogen	Symptoms	Intoxication/Infection	Diagnostic Test	Treatment
Vibrio parahaemolyticus Gastroenteritis Vibro parahaemolyticus Gastroenteritis	V. parahaemolyticus	Cholera-like diarrhea, but generally milder	Infection (enterotoxin)	Isolation of bacteria on 2-4% NaCl	Rehydration; antibiotics
Escherichia coli Gastroenteritis	EPEC, EIEC, EAEC, ETEC	Watery diarrhea	Infection (exotoxins)	Isolation on selective media, DNA fingerprinting	Oral rehydration
Shiga Toxin-Producing Enterohemorrhagic E. coli	E. coli O157:H7	Shigella -like dysentery; hemorrhagic colitis, HUS	Infection, Shiga toxin (exotoxin)	Isolation, sorbitol fermentation test, DNA fingerprinting	Intravenous rehydration, serum electrolyte monitoring
Campylobacter Gastroenteritis	Campylobacter jejuni	Fever, abdominal pain, diarrhea	Infection	Isolation in low O ₂ , high CO ₂	None
Helicobacter Peptic Ulcer Disease	Helicobacter pylori	Peptic ulcers	Infection	Urea breath test, bacterial culture	Antimicrobial drugs

Diseases in Focus 25.2 (3 of 3)

<i>Yersinia Gastroenteritis</i>	<i>Yersinia enterocolitica</i>	Abdominal pain and diarrhea, usually mild; may be confused with appendicitis	Infection (endotoxin)	Culture, serotyping	Oral rehydration
<i>Clostridium perfringens Gastroenteritis</i>	<i>Clostridium perfringens</i>	Usually limited to diarrhea	Infection (exotoxin)	Isolation of bacteria	Oral rehydration
<i>C. difficile-Associated Diarrhea</i>	<i>Clostridium difficile</i>	Mild diarrhea to colitis; 1–2.5% mortality	Infection (exotoxin)	Cytotoxin assay	Metronidazole, vancomycin
<i>Bacillus cereus Gastroenteritis</i>	<i>B. cereus</i>	May take form of diarrhea, nausea, vomiting	Intoxication	Isolation of ≥ 105 <i>B. cereus/g</i> food	None

Viral Diseases of the Digestive System

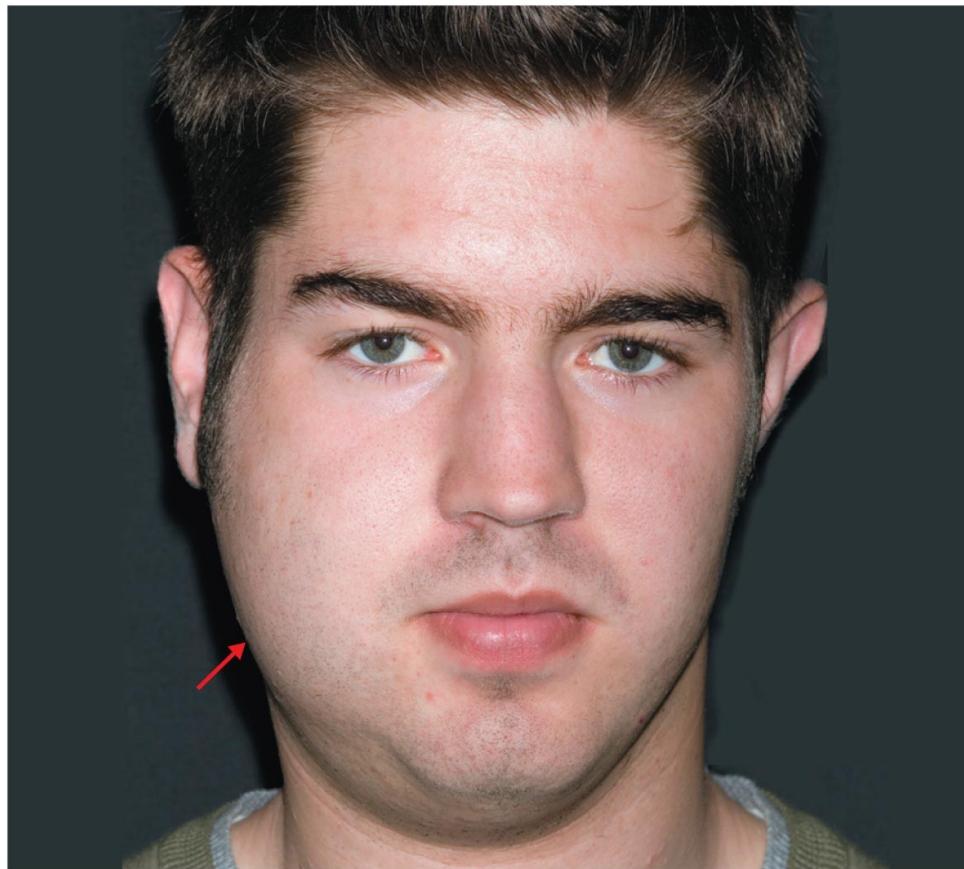
Learning Objectives

- 25-5 List the causative agents, modes of transmission, sites of infection, and symptoms for mumps.
- 25-6 Differentiate hepatitis A, hepatitis B, hepatitis C, hepatitis D, and hepatitis E.
- 25-7 List the causative agents, mode of transmission, and symptoms of viral gastroenteritis.

Mumps

- Virus targets the parotid glands
 - Painful swelling 16 to 18 days after exposure
- Transmitted in the saliva and respiratory secretions
 - Multiplies in respiratory tract and reaches the salivary glands via the bloodstream
- May cause orchitis (swelling of testes), meningitis, ovary inflammation, and pancreatitis
- Prevented with the MMR vaccine

Figure 25.13 A Case of Mumps



Check Your Understanding-5

Check Your Understanding

- ✓ Why is mumps included with the diseases of the digestive system?

25-5

Hepatitis

- Inflammation of the liver
- May result from drug or chemical toxicity, Epstein-Barr virus (EBV), cytomegalovirus (CMV), or the hepatitis viruses

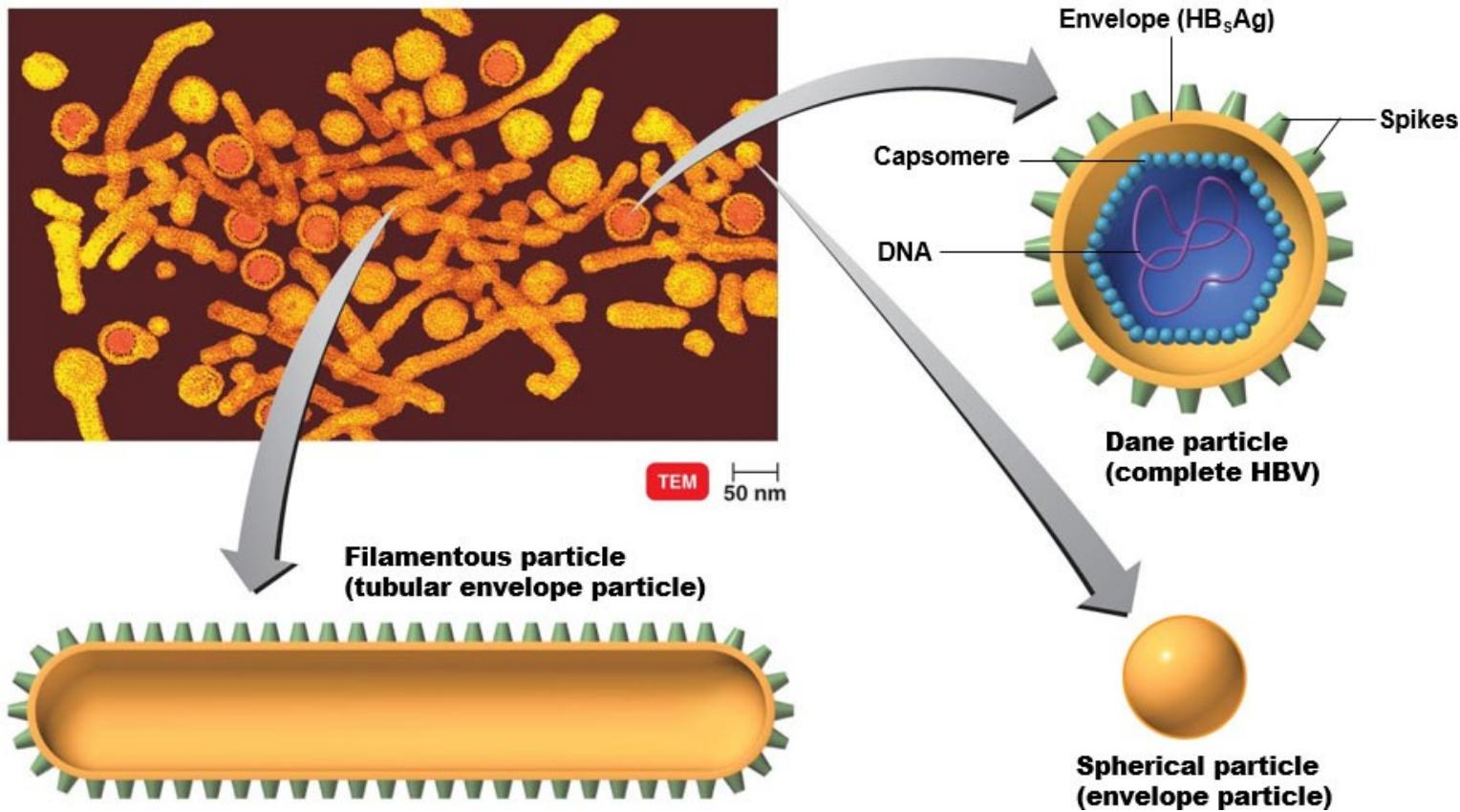
Hepatitis A

- Hepatitis A virus (HAV)
 - Single-stranded RNA; lacks an envelope
- Entry via the oral route; multiplies in the epithelial lining of the intestinal tract
 - Spreads to the liver, kidneys, and spleen
- Anorexia, malaise, nausea, diarrhea, fever, chills
 - Later, jaundice and dark urine
 - Symptoms last 2 to 21 days; low mortality
- Detected via IgM anti-HAV
- Treated with immune globulin
- Inactivated vaccine for prevention

Hepatitis B (1 of 2)

- Hepatitis B virus (HBV)
 - Double-stranded DNA; enveloped
 - Resembles retroviruses
- Transmitted via the blood and bodily fluids
 - Up to 1 billion viruses per ml of blood
 - Complete virions (Dane particle)
 - Spherical particles and filamentous particles
 - Lack DNA
 - Contain hepatitis B surface antigen (HB_sAg)
- Prevented by a vaccine
- Treated with hepatitis B immune globulin (HBIG)

Figure 25.14 Hepatitis B virus (HBV)



Hepatitis B (2 of 2)

- Acute hepatitis B
 - Often subclinical
 - Similar symptoms to hepatitis A; gradual recovery
 - **Fulminant hepatitis** in 1% of cases
 - Sudden massive liver damage; fatal
- Chronic hepatitis B (HB_eAg)
 - Involves 10% of infected patients
 - May lead to liver cirrhosis or liver cancer
 - Presence of HB_eAg indicates a vigorously replicating virus



Hepatitis C

- Hepatitis C virus (HCV)
 - Single-stranded RNA; enveloped
 - Often transfusion-transmitted
- Destroys the liver by using genetic variation to evade the immune response
- Kills more in the United States than AIDS
- 85% of cases become chronic
 - 25% develop liver cirrhosis or cancer
- Treated with peginterferon and ribavirin
- No vaccine

Diseases in Focus 25.3



Other Hepatitis Viruses

- **Hepatitis D**

- Acute (coinfection) and chronic (superinfection) forms
- Linked to hepatitis B infection

- **Hepatitis E (HEV)**

- Spread by fecal-oral transmission
 - Similar to hepatitis A
- Endemic in areas with poor sanitation

Characteristics of Viral Hepatitis

- After eating at one restaurant, 355 people were diagnosed with the same hepatitis virus.
- Can you identify infections that could cause these symptoms?

Diseases in Focus 25.3 (1 of 2)

Disease	Pathogen	Symptoms	Incubation Period	Method of Transmission	Diagnostic Test	Treatment	Vaccine
A	Hepatitis A virus, Picornaviridae	Mostly subclinical; fever, headache; malaise, jaundice in severe cases; no chronic disease	2–6 weeks	Ingestion	IgM antibodies	Immunoglobulin	Inactivated virus. Post-exposure immune Globulin
B	Hepatitis B virus, Hepadnaviridae	Frequently subclinical; similar to HAV, but no headache; more likely to progress to severe liver damage; chronic disease occurs	4–26 weeks	Parenteral; sexual contact	IgM antibodies	Interferon alfa and nucleoside analogue s	Genetically modified vaccine produced in yeast

Diseases in Focus 25.3 (2 of 2)

Disease	Pathogen	Symptoms	Incubation Period	Method of Transmission	Diagnostic Test	Treatment	Vaccine
C	Hepatitis C virus, Flaviviridae	Similar to HBV, more likely to become chronic	2–22 weeks	Parenteral	PCR for viral RNA	Peginterferon and ribavirin	None
D	Hepatitis D virus, Deltaviridae	Severe liver damage; high mortality rate; chronic disease may occur	6–26 weeks	Parenteral; requires coinfection with hepatitis B	IgM antibodies	None	HBV vaccine is protective
E	Hepatitis E virus, Caliciviridae	Similar to HAV, but pregnant women may have high mortality; no chronic disease	2–6 weeks	Ingestion	IgM antibodies, PCR for viral RNA	None	HAV vaccine is protective

Check Your Understanding-6

Check Your Understanding

- ✓ Of the several hepatitis diseases, HAV, HBV, HCV, HDV, and HEV, which two now have effective vaccines to prevent them?

25-6

Viral Gastroenteritis

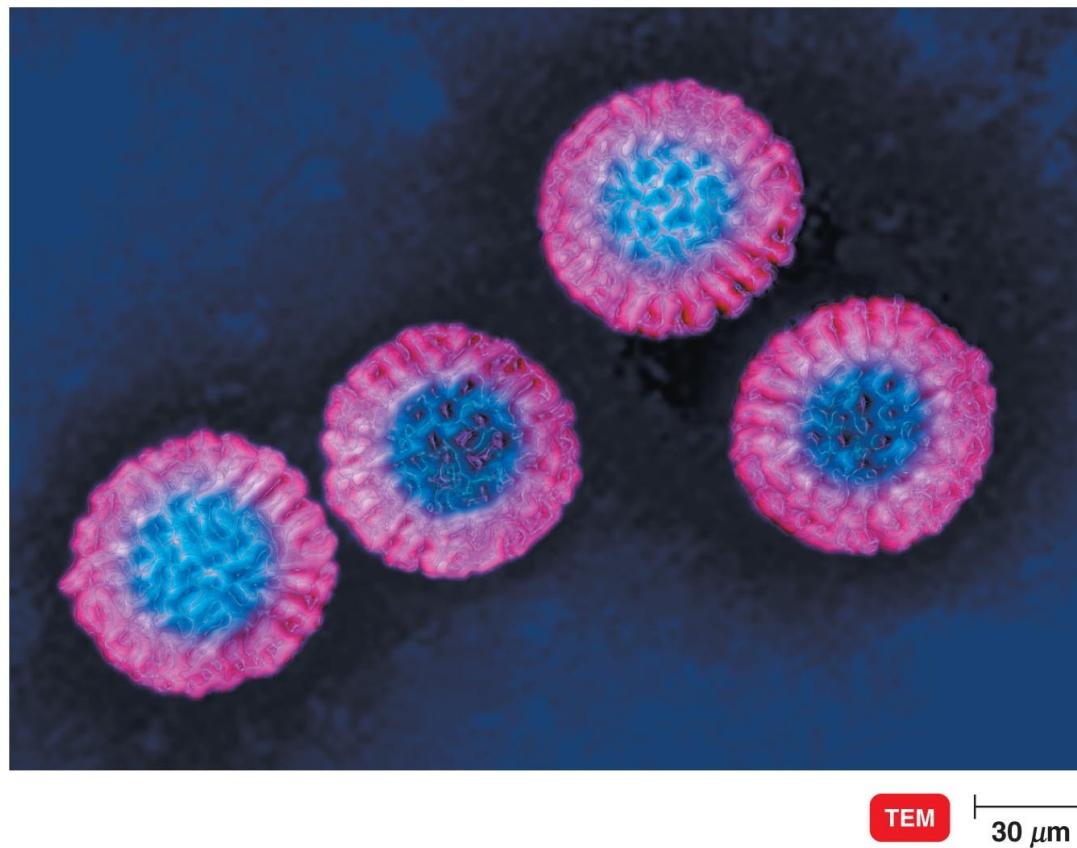
- **Rotavirus**

- Common in children; low mortality
- 2 to 3 day incubation; low-grade fever, diarrhea, vomiting
- Prevented with a live oral vaccine

- **Norovirus**

- Caliciviruses
- Fecal-oral transmission
- Low infectious dose
- 18 to 48 hour incubation; diarrhea and vomiting
- Detected with PCR and EIA tests

Figure 25.15 Rotavirus



Check Your Understanding-7

Check Your Understanding

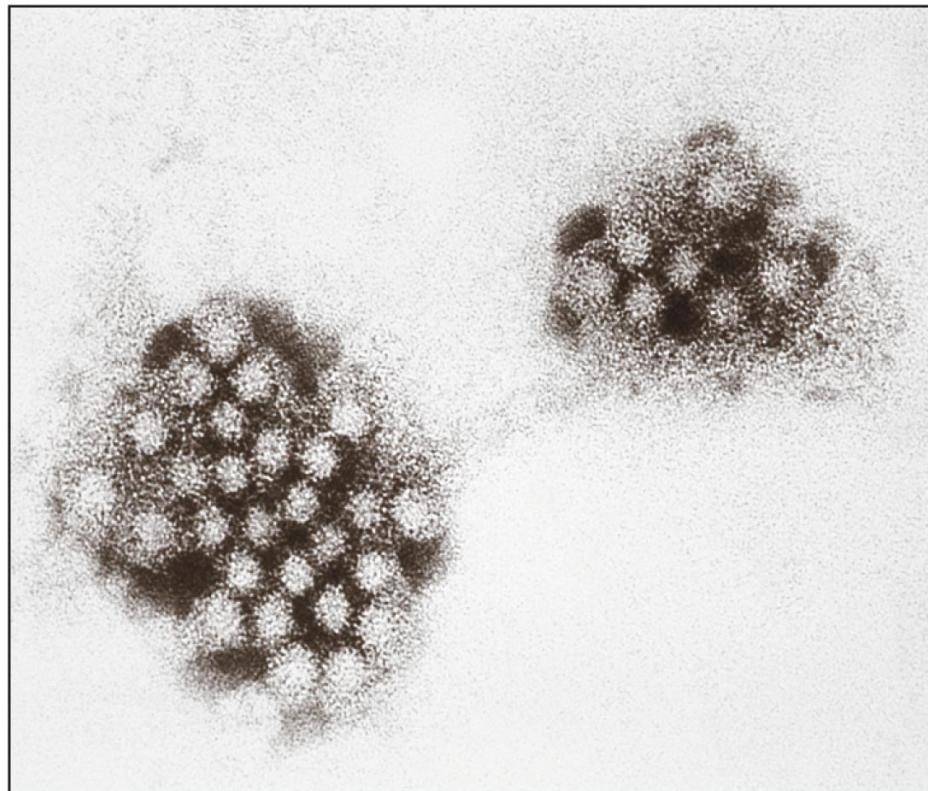
- ✓ Two very common causes of viral gastroenteritis are caused by rotaviruses and noroviruses. Which of these now can be prevented by a vaccine?

25-7

Diseases in Focus: Viral Diseases of the Digestive System

- An outbreak of diarrhea begins in mid-June, peaks in mid-August, and tapers off in September. A clinical case is defined as diarrhea (three loose stools during a 24-hour period) in a member of a swim club. The virus shown is isolated from one patient.
- Can you identify infections that could cause these symptoms?

Diseases in Focus 25.4 (1 of 2)



TEM
50 nm

Diseases in Focus 25.4 (2 of 2)

Disease	Pathogen	Symptoms	Incubation Period	Diagnostic Test	Treatment
Mumps	Mumps virus, Paramyxoviridae	Painful swelling of parotid glands	16–18 days	Symptoms; virus culture	Preventive vaccine
Viral Gastroenteritis	Rotavirus	Vomiting, diarrhea for 1 week	1–3 days	Enzyme immunoassay for viral antigens in feces	Oral rehydration
	Norovirus	Vomiting, diarrhea for 2–3 days	18–48 hr	PCR	Oral rehydration

Fungal Diseases of the Digestive System (1 of 2)

Learning Objective

25-8 Identify the causes of ergot poisoning and aflatoxin poisoning.

Fungal Diseases of the Digestive System (2 of 2)

- Mycotoxins: toxins produced by fungi
 - Blood diseases
 - Nervous system disorders
 - Kidney damage
 - Liver damage
 - Cancer

Ergot and Aflatoxin Poisoning

- **Ergot poisoning**
 - Mycotoxins produced by **Claviceps purpurea**
 - Occurs in grains
 - Restricts blood flow (gangrene) and causes hallucinations
- **Aflatoxin poisoning**
 - Mycotoxins produced by **Aspergillus flavus**
 - Likely to be found on peanuts
 - Causes liver cirrhosis and liver cancer

Check Your Understanding-8

Check Your Understanding

- ✓ What is the connection between the occasional hallucinogenic symptoms produced by ergot poisoning and a modern illicit drug?
25-8

Protozoan Diseases of the Digestive System

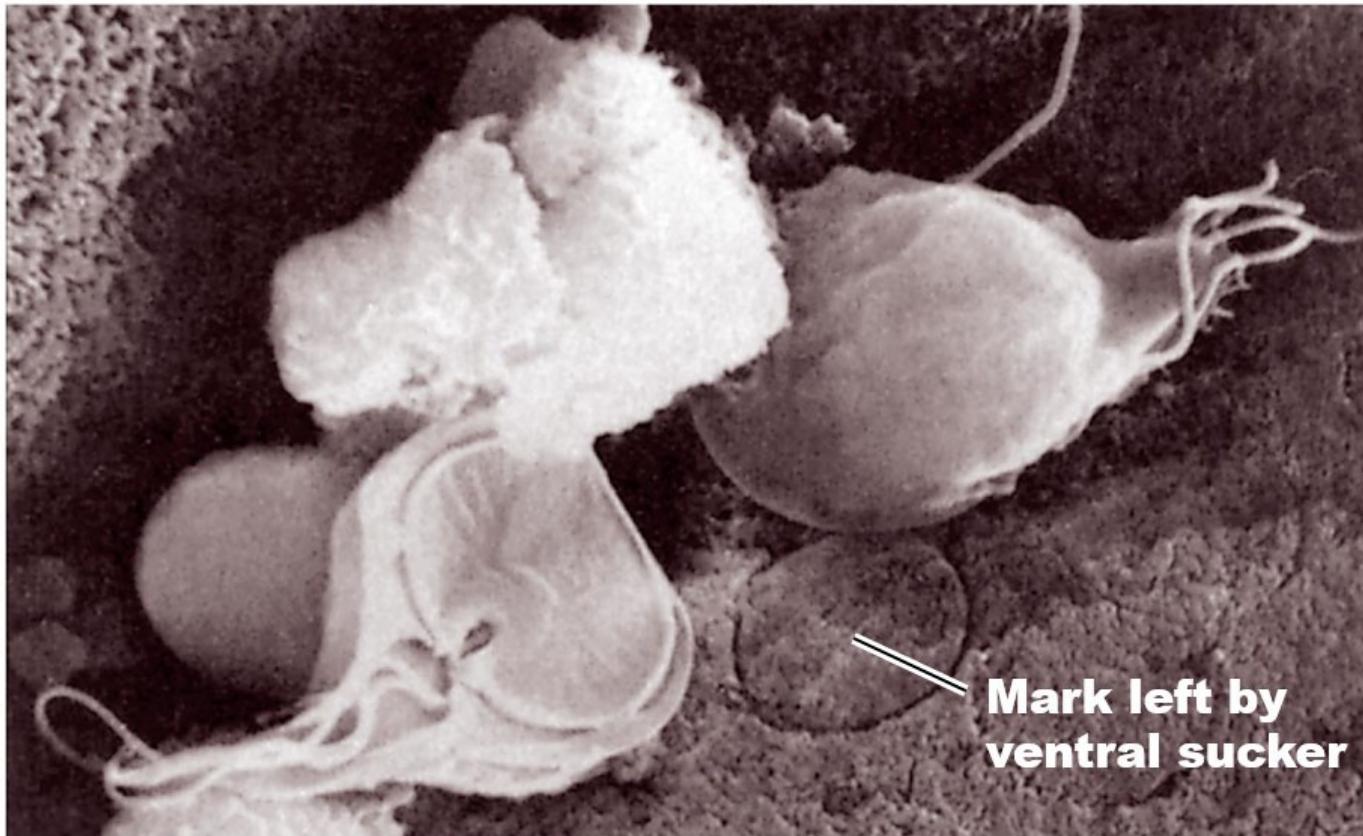
Learning Objective

25-9 List the causative agents, modes of transmission, symptoms, and treatments for giardiasis, cryptosporidiosis, **Cyclospora** diarrheal infection, and amebic dysentery.

Giardiasis

- Caused by **Giardia intestinalis**
 - Flagellated protozoan
 - Forms cysts in feces and water; trophozoites in the body
 - Attaches to the intestinal wall
- Prolonged diarrhea, malaise, weight loss, flatulence, cramps
 - Hydrogen sulfide detected in the breath or stools
- Diagnosed with a string test, ELISA, or FA test
- Treated with metronidazole and nitazoxanide

Figure 25.16 The trophozoite form of *Giardia intestinalis*, the flagellated protozoan that causes giardiasis



SEM

5 μm

Check Your Understanding-9

Check Your Understanding

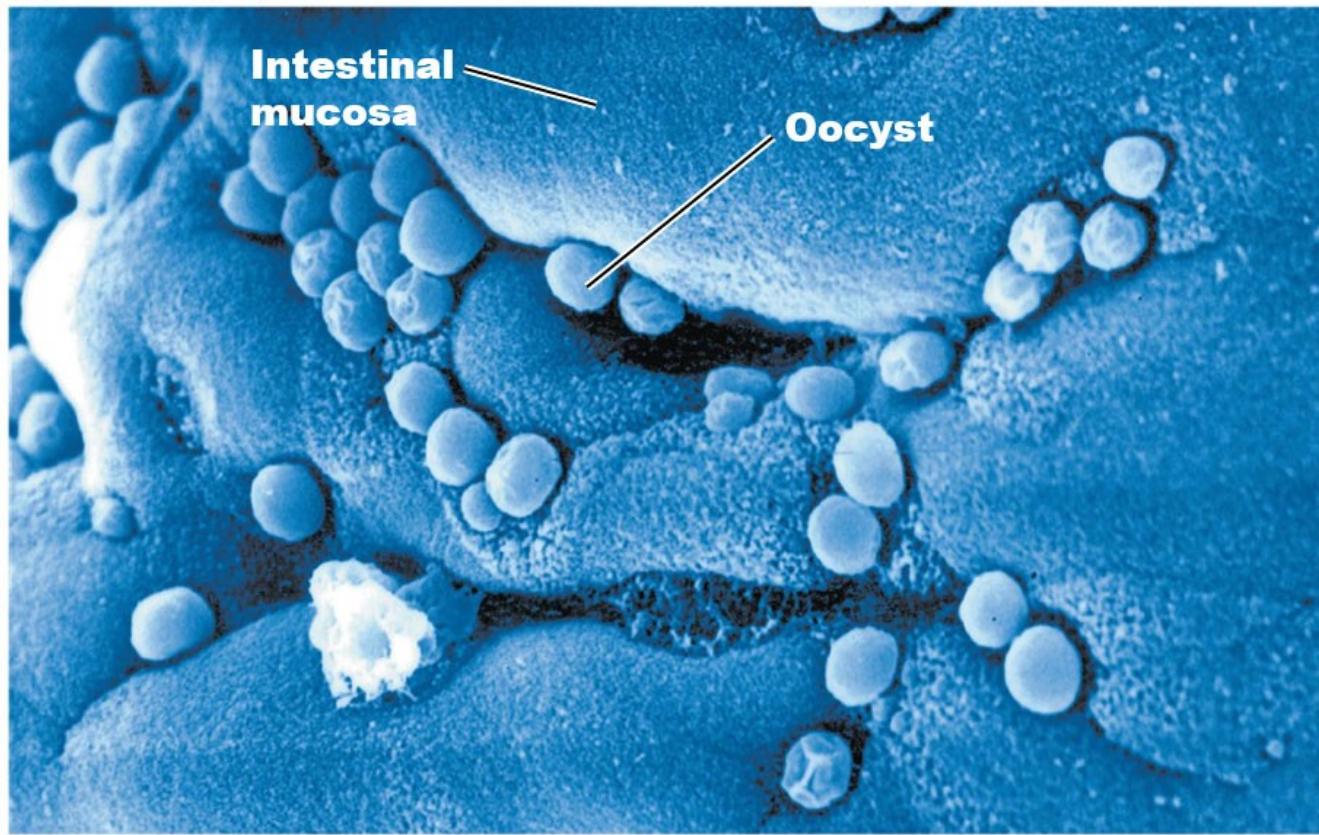
- ✓ Is giardiasis caused by ingestion of a cyst or an oocyst?

25-9

Cryptosporidiosis

- Caused by **Cryptosporidium parvum** and **C. hominis**
- Ingested oocysts release sporozoites
 - Invade the intestinal epithelium
- Cholera-like diarrhea for 10 to 14 days
- Transmitted through drinking water
 - Resistant to chlorination
- Diagnosed with an FA test or immunoassay tests
- Treatment with nitazoxanide

Figure 25.17 Cryptosporidiosis



SEM
5 μm

Cyclospora Diarrheal Infection

- Caused by **Cyclospora cayetanensis**
- Days or weeks of watery diarrhea
- Oocysts are ingested from drinking water contaminated with feces
- No satisfactory diagnostic tests
- Treatment with trimethoprim and sulfamethoxazole

Amebic Dysentery (Amebiasis)

- Caused by **Entamoeba histolytica**
 - Produces cysts that survive stomach acid
 - Trophozoites produced from cysts in the intestines
 - Multiply in the wall of the large intestine
- Feces contain blood and mucus
- Can perforate the intestinal wall, causing abscesses
 - Organisms invade the liver
- Detection with latex agglutination and FA tests

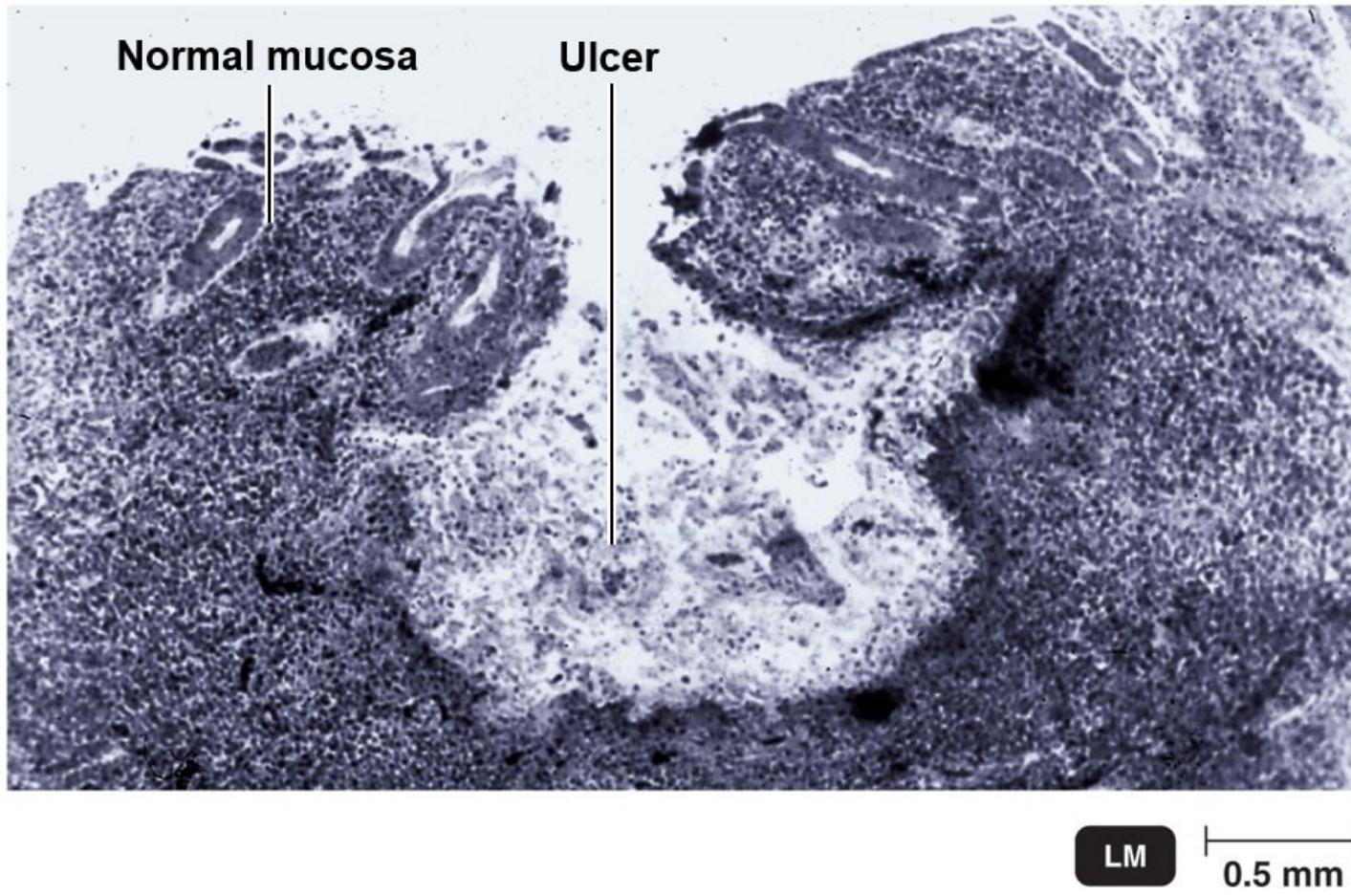


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Treatment with metronidazole plus Iodoquinol

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Figure 25.18 Section of Intestinal Wall Showing a Typical Flaskshaped Ulcer Caused by *Entamoeba histolytica*

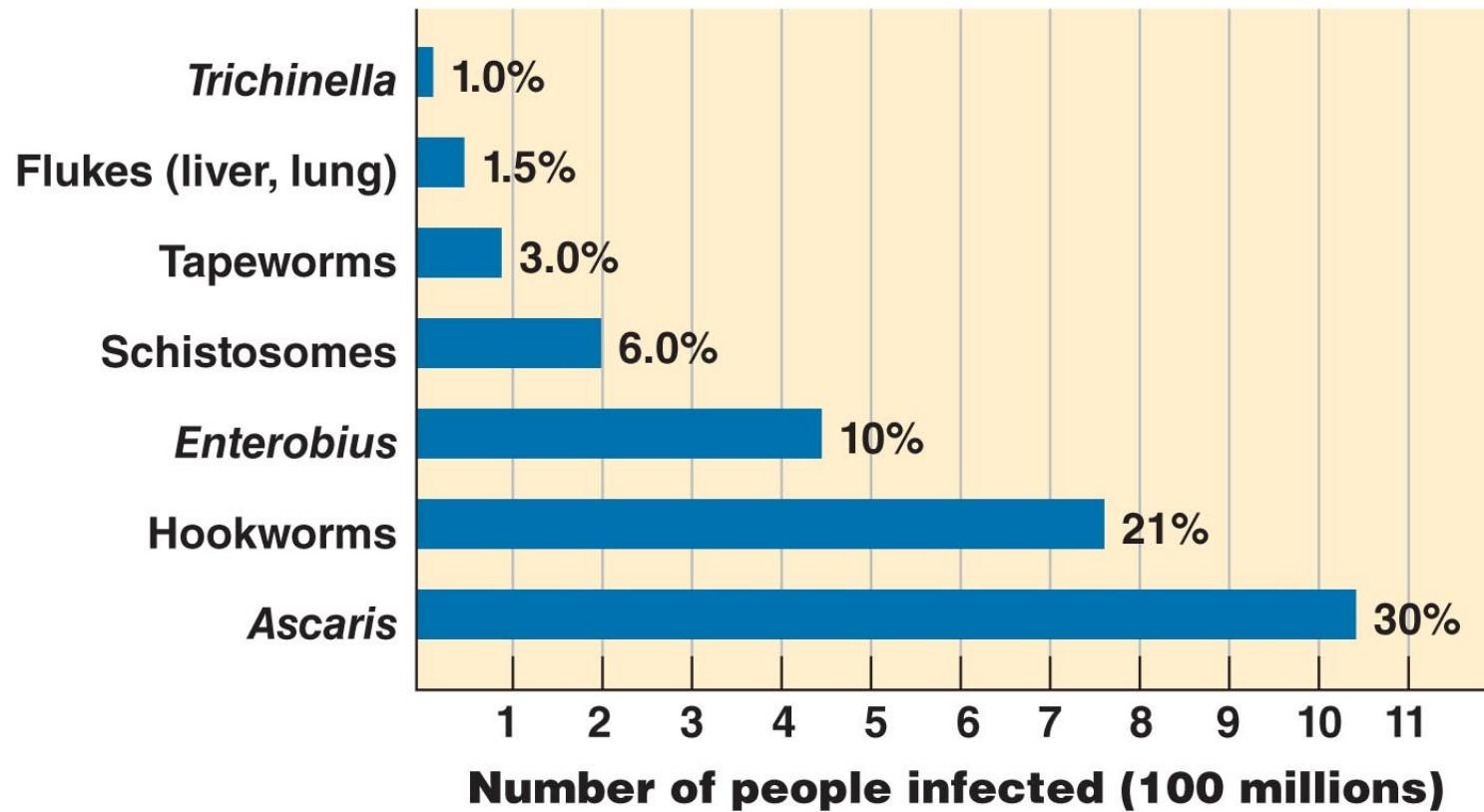


Helminthic Diseases of the Digestive System

Learning Objective

25-10 List the causative agents, modes of transmission, symptoms, and treatments for tapeworms, hydatid disease, pinworms, hookworms, whipworms, ascariasis, and trichinellosis.

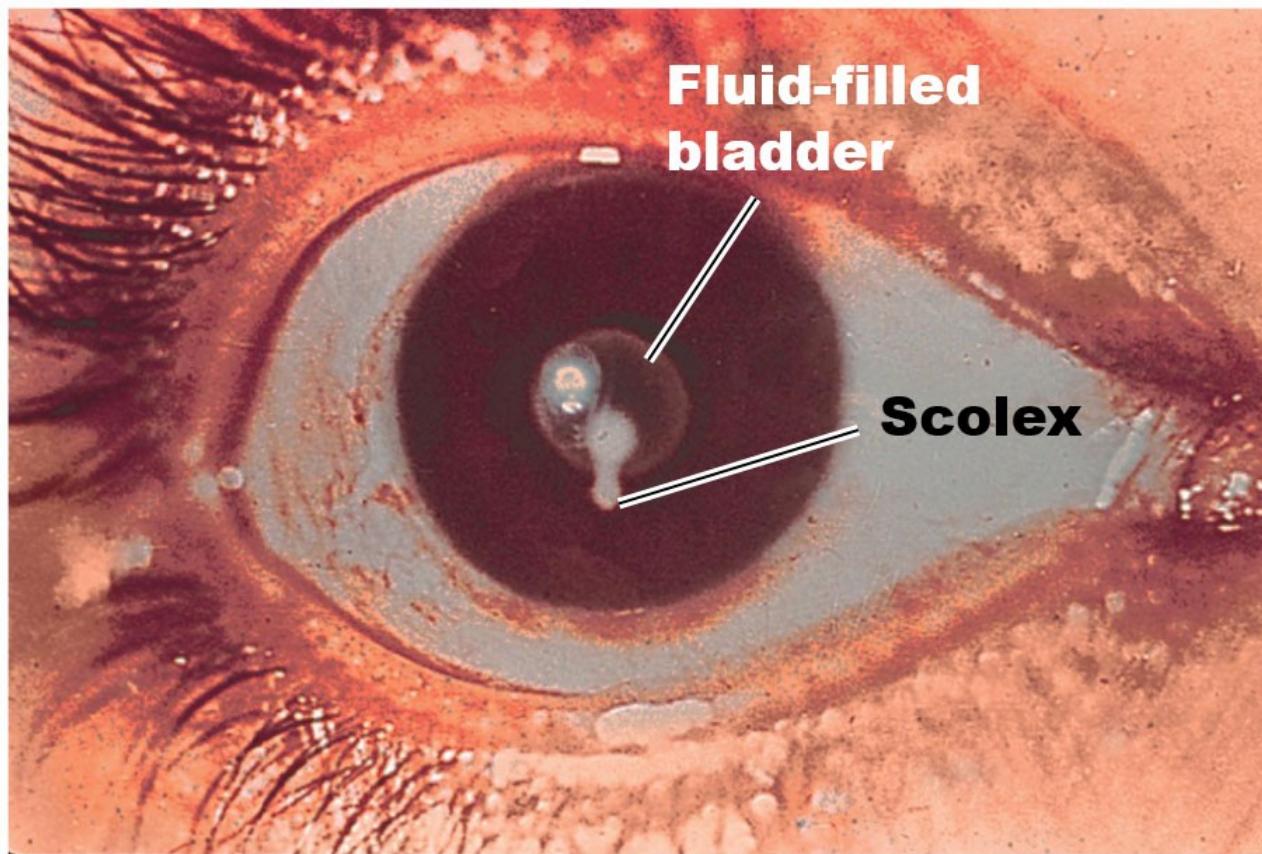
Figure 25.19 The Worldwide Prevalence of Human Infections with Selected Intestinal Helminths



Tapeworms (1 of 2)

- Beef tapeworm: **Taenia saginata**
- Pork tapeworm: **Taenia solium**
- Fish tapeworm: **Diphyllobothrium latum**
- **Taeniasis:** adult tapeworm infects the intestine
- **Cysticercosis:** infection with the larval stage by ingesting eggs
- **Ophthalmic cysticercosis:** larvae lodge in the eye
- **Neurocysticercosis:** larvae develop in the central nervous system

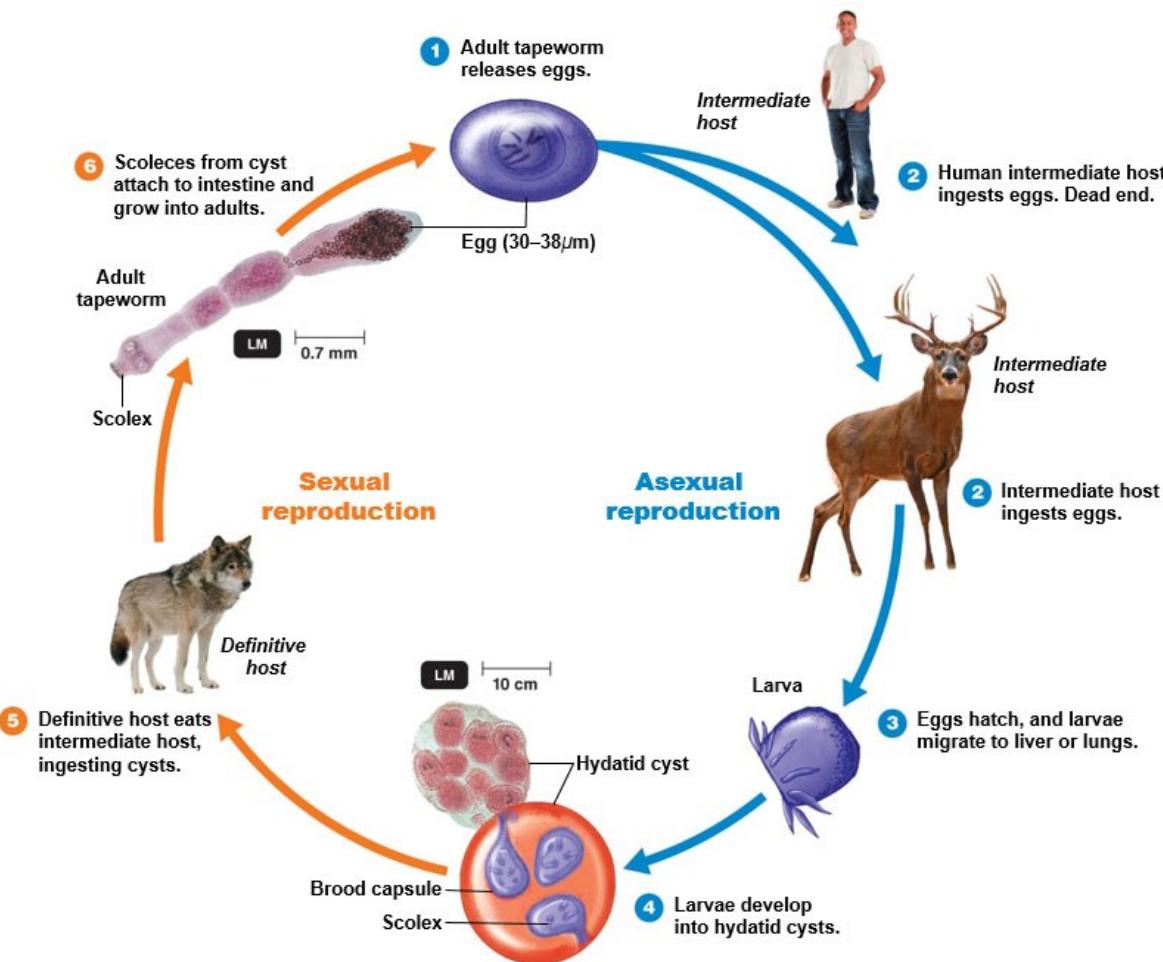
Figure 25.20 Ophthalmic Cysticercosis



Tapeworms (2 of 2)

- Three-stage life cycle
 - Eggs are excreted in the feces and ingested by animals
 - Eggs hatch into a larval cysticercus that lodges in the muscle
 - Human ingests undercooked meat containing cysticerci, which develop into adult tapeworms in the intestine
- Diagnosis with eggs or segments in the feces
- Treatment with praziquantel and albendazole

Figure 12.28 The Life Cycle of the Tapeworm, *Echinococcus* spp



Check Your Understanding-10

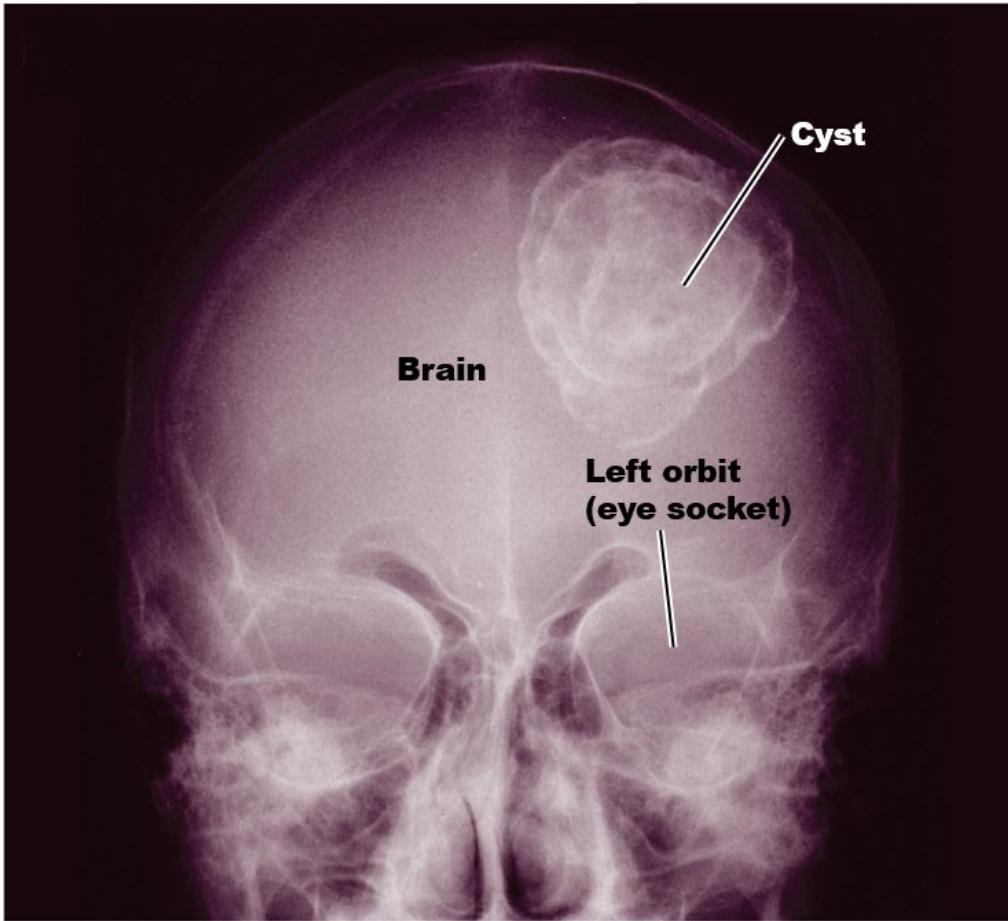
Check Your Understanding

- ✓ What species of tapeworm is the cause of cysticercosis?
25-10

Hydatid Disease

- Caused by the tapeworm **Echinococcus granulosus**
- Eggs are ingested and migrate to the liver, lungs, or brain
 - Develops a **hydatid cyst**
 - Can grow and hold up to 15 liters of fluid
 - May rupture, causing anaphylactic shock
- Diagnosis with serological tests, X rays, CT, and MRI
- Treatment with surgical removal or albendazole

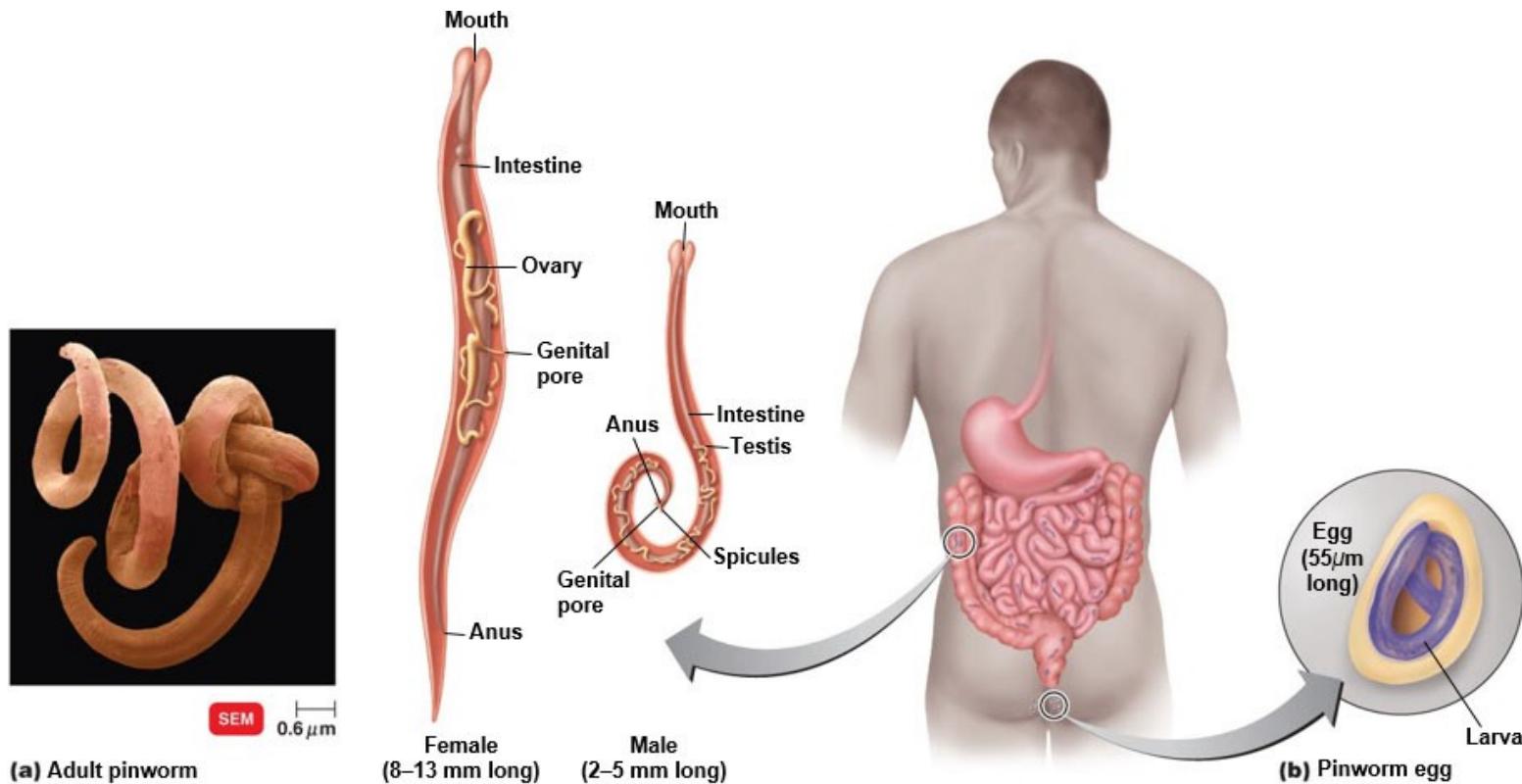
Figure 25.21 A Hydatid Cyst Formed by Echinococcus Granulosus



Pinworms

- **Enterobius vermicularis**
 - Tiny nematode
- Lays eggs around the anus, causing local itching
- Treatment with pyrantel pamoate and mebendazole

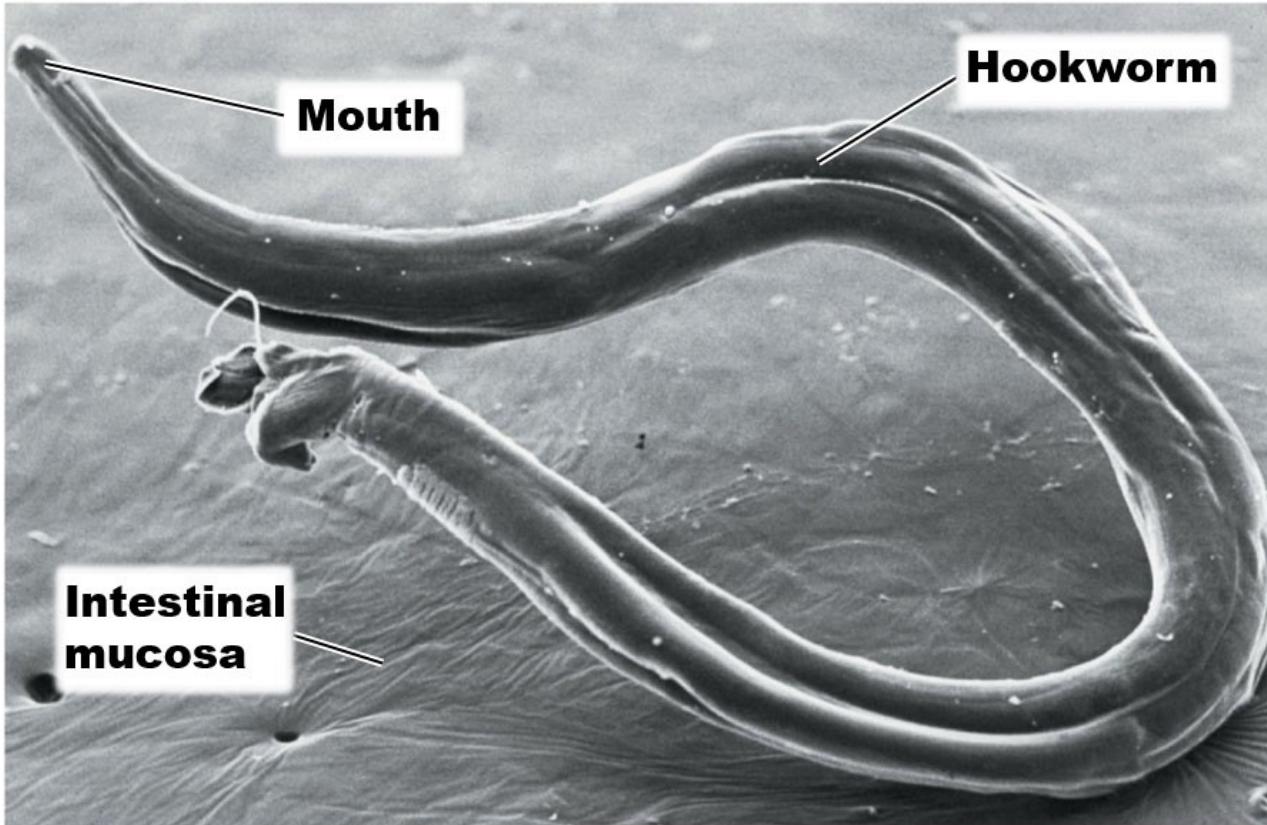
Figure 12.29 The Pinworm, *Enterobius Vermicularis*



Hookworms

- Caused by the nematodes **Necator americanus** and **Ancylostoma duodenale**
- Attaches to the intestinal wall and feeds on blood and tissue
- Anemia, lethargic behavior, and craving for peculiar foods (pica)
- Carried from human feces in soil that contact bare skin
- Treatment with mebendazole

Figure 25.22 Ancylostoma Hookworm



SEM 0.6 mm

Ascariasis

- Caused by the nematode **Ascaris lumbricoides**
- 30% of the worldwide population is infected
- Eggs shed in the feces and are ingested by another person
 - Hatch into larvae and pass into the bloodstream and lungs
 - Larvae migrate to the throat and are swallowed
 - Larvae develop into adult worms in the intestinal tract and emerge from the anus, mouth, or nose

Figure 25.25 *Ascaris Lumbricoides*, the Cause of Ascariasis



Whipworm (*Trichuris trichiura*)

- 30 to 50 mm in length
- Distribution similar to **A. lumbricoides**
- Eggs are ingested and enter the intestinal glands
 - Eggs hatch and grow on the intestinal surface
 - Adult worm buries into the intestinal mucosa and lives as a parasite
 - Feeds on cell contents and blood
- Causes anemia, malnutrition, and retarded growth

Figure 25.24 Egg of *Trichuris trichiura*

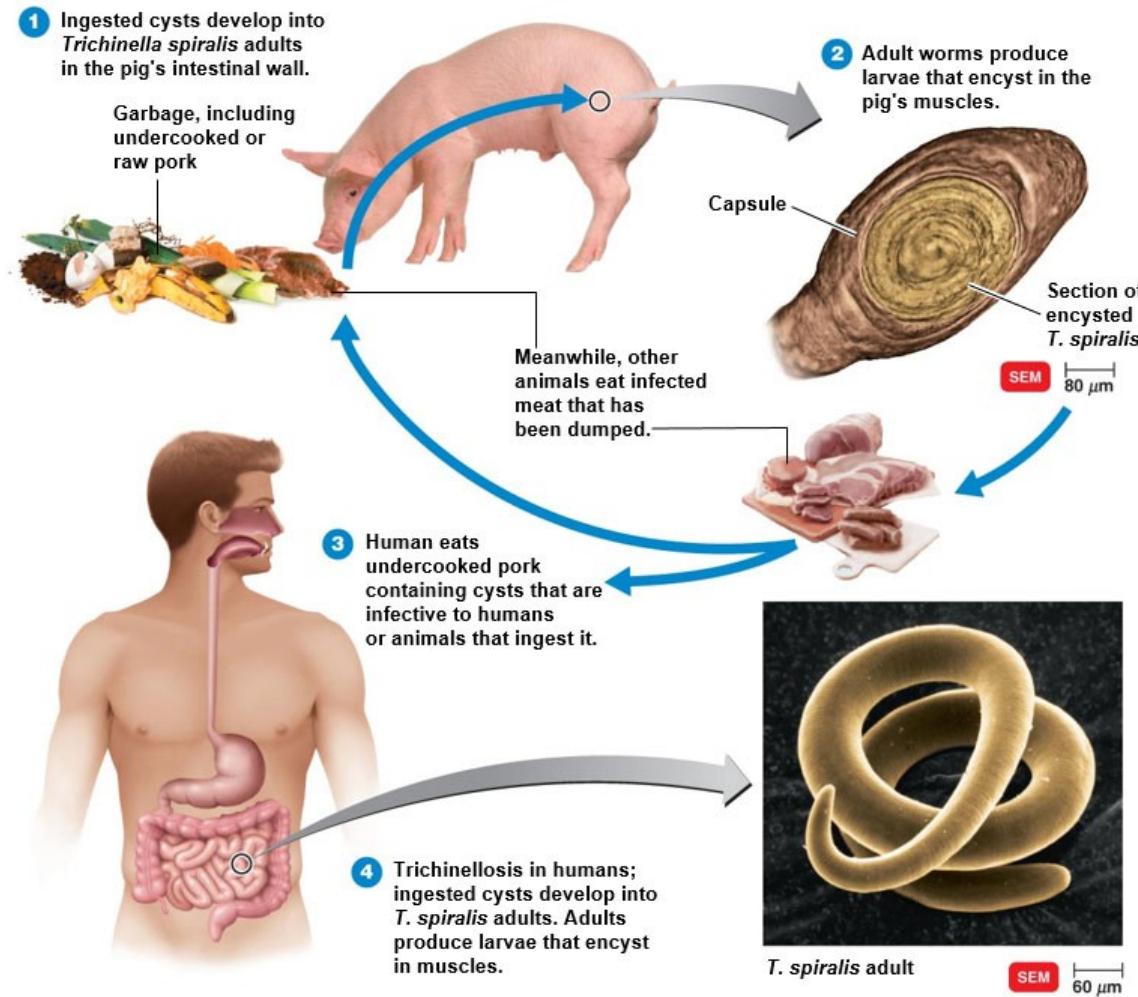


LM 10 μm

Trichinellosis

- Caused by **Trichinella spiralis**
- Encysted larvae are ingested from undercooked pork and other meats
 - Digestion removes the cyst wall and the worm matures into an adult
 - Adults produce larvae in the intestines that invade tissues and form new cysts
- Fever, eye swelling, gastrointestinal upset
- Treatment with albendazole or mebendazole

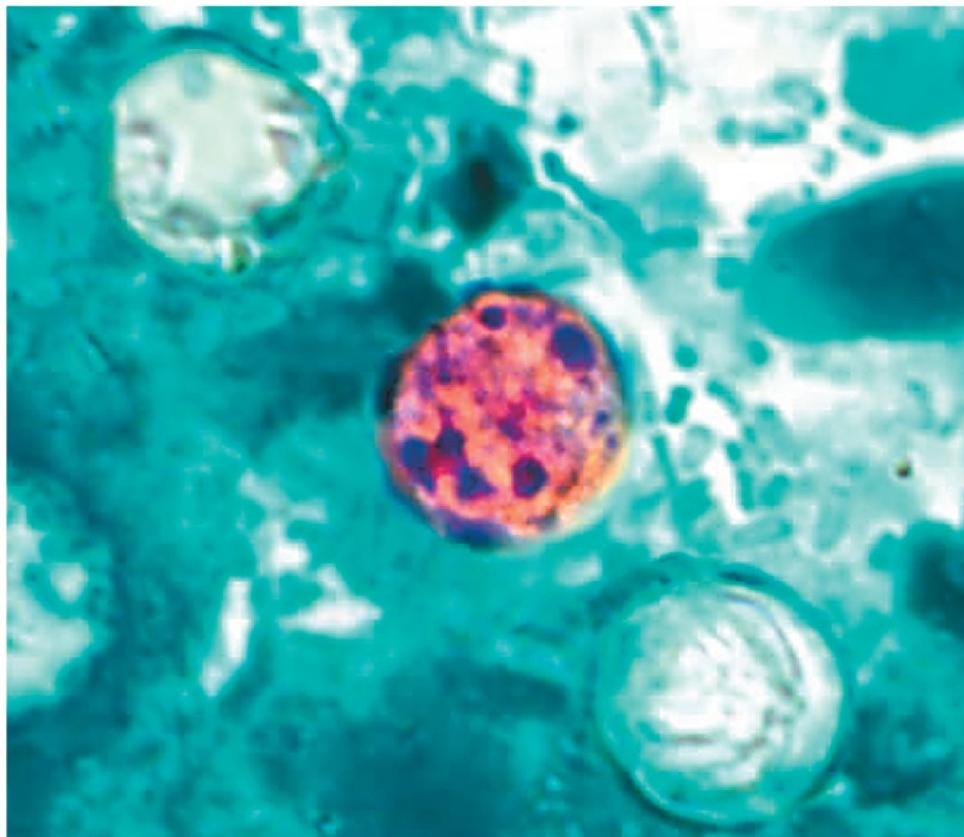
Figure 25.25 The Life Cycle of *Trichinella spiralis*, the Causative Agent of Trichinellosis



Diseases in Focus: Fungal, Protozoan, and Helminthic Diseases of the Lower Digestive System

- Public health officials in Pennsylvania are notified of cases of watery diarrhea with frequent, sometimes explosive, bowel movements among persons associated with a residential facility (e.g., residents, staff, and volunteers). The disease is associated with eating snow peas.
- Can you identify infections that could cause these symptoms?

Diseases in Focus 25.5 (1 of 4)



LM 
3 μm

Diseases in Focus 25.5 (2 of 4)

Disease	Pathogen	Symptoms	Reservoir or Host	Diagnostic Test	Treatment
Fungal Diseases					
Ergot Poisoning	<i>Claviceps purpurea</i>	Restricted blood flow to limbs; hallucinogenic.	Mycotoxin produced by fungus growing on grains	Finding fungal sclerotia in food	None
Aflatoxin Poisoning	<i>Aspergillus flavus</i>	Liver cirrhosis; liver cancer.	Mycotoxin produced by fungus growing on food	Immunoassay for toxin in food	None
Protozoan Diseases					
Giardiasis	<i>Giardia intestinalis</i>	Protozoan adheres to intestinal wall, may inhibit Nutritional absorption; diarrhea.	Water; mammals	FA	Metronidazole; quinacrine
Cryptosporidiosis	<i>Cryptosporidium hominis, C. parvum</i>	Self-limiting diarrhea; may be life-threatening in immunosuppressed	Cattle; water	Acid-fast stain; FA; ELISA	Oral rehydration

Diseases in Focus 25.5 (3 of 4)

Disease	Pathogen	Symptoms	Reservoir or Host	Diagnostic Test	Treatment
Cyclospora Diarrheal Infection	<i>Cyclospora cayetanensis</i>	Watery diarrhea.	Humans; birds; usually ingested with fruits and vegetables	Acid-fast stain	Trimethoprim and sulfamethoxazole
Amebic Dysentery (amebiasis)	<i>Entamoeba histolytica</i>	Ameba lyses epithelial cells of intestine, causes abscesses; significant mortality rate.	Humans	Microscopy; serology	Metronidazole
Helminthic Diseases					
Tapeworms	<i>Taenia saginata, T. solium, Diphyllobothrium latum</i>	Helminth leaves few symptoms; pork tapeworm larvae may encyst in many organs (neurocysticercosis) and cause damage.	Intermediate host: cattle, pigs, fish; definitive host: humans	Microscopic exam of feces	Praziquantel; albendazole
Hydatid Disease	<i>Echinococcus granulosus</i>	Larvae form in body; may be very large and	Intermediate host: humans;	Serology; X-ray exam	Surgical removal; albendazole

Diseases in Focus 25.5 (4 of 4)

Disease	Pathogen	Symptoms	Reservoir or Host	Diagnostic Test	Treatment
Pinworms	<i>Enterobius vermicularis</i>	Itching around anus.	Intermediate and definitive hosts: humans	Microscopic exam	Pyrantel pamoate
Hookworms	<i>Necator americanus, Ancylostoma duodenale</i>	Large infections may result in anemia.	Larvae enter skin from soil; definitive hosts: humans	Microscopic exam	Mebendazole
Ascariasis	<i>Ascaris lumbricoides</i>	Helminths live off undigested intestinal contents, causing few symptoms.	Intermediate and definitive hosts: humans	Microscopic exam	Mebendazole
Whipworm	<i>Trichuris trichiura</i>	Diarrhea, malnutrition.	Intermediate and definitive hosts: humans	Microscopic exam of feces	Albendazole, mebendazole
Trichinellosis	<i>Trichinella spiralis, T. nativa</i>	Larvae encyst in striated muscle; usually few symptoms, but large infections may be fatal.	Intermediate and definitive hosts: mammals (including humans)	Biopsy; ELISA	Mebendazole ; corticosteroids